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THE UNIVERSITY OF ALBERTA

NEUROPSYCHOLOGICAL CHARACTERISTICS OF HALLUCINATED PSYCHIATRIC PATIENTS

BY

DAVID MUNRO GILL

(C)

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE
OF.......DOCTOR OF PHILOSOPHY.............................
IN
COUNSELLING PSYCHOLOGY
DEPARTMENT OF EDUCATIONAL PSYCHOLOGY

EDMONTON, ALBERTA

(SPRING 1989)

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Abstract

The purpose of this case control investigation was to examine the neuropsychological sequelae of hallucinations in hospitalized psychiatric patients. A battery of twelve neuropsychological tests plus the Wechsler intelligence scales was administered to a sample of patients and controls. 343 cases were assessed (182 controls, 161 patients) and because of non-normality and distributional issues all data were converted to ranks before further analysis.

Control subjects were rigorously screened for neurological disorders and patients belonged either to a strictly diagnosed group (selected, n=54) or a heterogeneous group (unselected, n=107). Assessment of hallucinations was by examination of the case notes of the psychiatrists who established the patient's diagnosis (selected sample) or by a chart review of the patient (unselected sample).

It was hypothesized that there would be differences between patients and controls, hallucinators and non-hallucinators, and between selected and non-selected hallucinators. Results of analyses showed clear neuropsychological differences between all patient groups and controls, but not between hallucinators and non-hallucinators.

A severity dimension emerged in which unselected non-hallucinators showed less brain dysfunction than unselected hallucinators. They in turn were less impaired then selected non-hallucinators who were similar to selected hallucinators. Findings were explained in terms of the concepts of sensitivity and specificity, in that neuropsychological measures were sensitive to the brain dysfunction found in psychopathological groups, but ~~were~~ not specific to discrete symptoms within those groups.

Supplementary analysis established a factor structure for patients and controls and examined the intercorrelations between the Wechsler scales and the neuropsychological tests. Neuropsychological tests predicted more Wechsler scale score variance than Wechsler scales predicted neuropsychological test variance. In order to more efficiently investigate symptom variables such as hallucinations it was proposed that neuropsychological and electrophysiological techniques be combined in order to assess both cortical and subcortical aspects of hallucinations.

Acknowledgements

I would like to thank the following people who made completion of this dissertation possible. My advisor, Dr. Henry Janzen who provided both the necessary direction and freedom required for this investigation. Dr. John Reddon whose steady encouragement and constructive criticism so ably shaped the entire project. My committee members, Dr. David Ho, Dr. Robert Short, Dr. Einer Boberg, Dr. John Paterson, and Dr. Lorne Yeudall who continues to help me think more critically about human brain function. The staff of the Department of Neuropsychology at Alberta Hospital Edmonton deserve special mention for their logistic and technical assitance.

Lastly, but by no means last, I wish to express my gratitude to my wife, Penny, and to my family for their patience and unwavering support throughout all the phases of this dissertation.

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Chapter I

General Hallucinations

1.1 Synopsis of Chapter

This chapter introduces the concept of hallucinations and provides background information concerning the use of neuropsychological measures to assess hallucinations in the mentally ill. Definitional and classification issues are briefly discussed and a methodological overview of the investigation is provided. Scope and limitations of the study are described. Finally, a brief outline of the dissertation is provided.

1.2 Significance of the Dissertation

> The mentally ill have always been with us - to be feared, marveled at, laughed at, pitied, or tortured, but all too seldom cured. Their existence shakes us to the core of our being, for they make us painfully aware that sanity is a fragile thing. To cope with their ills man has always needed a science that could penetrate to where the natural sciences cannot probe - into the universe of man's mind (Alexander & Selesnick, 1966, p. 3).

Psychiatry, the branch of medicine that studies mental illness, has existed in varying forms since before the time of the ancient Greeks (Alexander & Selesnick, 1966). And while it is generally accepted that hallucinations are an important symptom that helps to define mental illness, just what exactly constitutes an illness or a disease is still problematic. Beyond being that which physicians treat, there is no commonly accepted, rigorous definition of disease (see Kendell, 1975 and Merskey, 1986 for a discussion of the concept of disease). However, the association of hallucinations with disease dates back to the Middle Ages (Johnson, 1978).

Hallucinations, as studied in this dissertation, are those which occur in a context of mental illness and are viewed as symptoms of psychopathology. The methods of investigation are those of clinical neuropsychology, a relatively recent branch of Psychology which is concerned with the relationship between brain dysfunction or damage, and behavior (Crockett, Clark, & Klonoff, 1981; Davison, 1974; Sacks, 1987). The brain is viewed as the organ of behavior and hallucinations are conceptualized as the result of cerebral processes. Within the field of psychiatry, the techniques of clinical neuropsychology are being applied to psychopathological syndromes (Flor-Henry, in press b) and it seems reasonable to extend this approach to the study of specific symptoms (Persons, 1986).

It is argued that the hallucinations experienced by those people suffering from a mental illness are due to abnormal brain functioning and that the techniques of clinical neuropsychology are a sensitive measure of cerebral dysfunction. Hallucinations are a distressing experience that can, in some cases, be severe enough to cause the person so afflicted to commit acts of violence or to take their own life (Hellerstein, Frosch, & Koenigsberg, 1987). Improved understanding and delineation of the cerebral mechanisms of hallucinations is one way to improve both the psychological and pharmacological treatment of these stressful and sometimes fatal mental experiences.

1.3 Background of Hallucinations

Historically, hallucinations are a ubiquitous phenomena (Bourguignon, 1970; Wallace, 1959). Visions and voices as a part of the individual's mental experience can be traced back to the time of the ancient Greeks (Johnson, 1978; Slade, 1976c). Derived from the Latin verb "allucinatio," meaning wandering of the mind, idle talk or prating, hallucinations have in recent years come to be intimately associated with mental illness (Asaad & Shapiro, 1986; Fish, 1976; Schneider, 1959; Walker & Cavenar, 1983).

1.3.1 Historical Overview of Hallucinations

Socrates (469-399 B.C.) is described as having experienced auditory hallucinations, which he regarded as sources of divine inspiration. The voices, present since childhood, functioned to guide his behavior by forbidding him to do certain things (Johnson, 1978). Aristotle (348-322 B.C.) is also reported to have experienced hallucinations and the physician Asclepiades, who practiced in the first century B.C., provided the first clinical differentiation between delusions and hallucinations. This distinction was later popularized by Esquirol in the early nineteenth century (Zilboorg & Henry, 1941).

The Greeks saw a commonality between the experiences of dreaming and hallucinating. Both Plato (427-348 B.C.) and Aristotle commented on this and Aristotle suggested that a similar process was responsible for both states (Evarts, 1962). At this time hallucinations were not related to mental illness, but were viewed as being caused by external forces. They were considered to be potential sources of insight into the future and those experiencing hallucinations were neither persecuted nor stigmatized (Zilboorg & Henry, 1941). It should be noted, however, that the Greeks were cognizant of mental illness. Their astute description of clusters of behaviors are still recognizable today as the syndromes of hysteria, dementia, depression, mania, anxiety states, phobias, epilepsy, obsessions, and schizophrenia (Moss, 1967).

With the cultural ascendancy of Rome, official policy towards hallucinists changed because of the Roman emperors' fears that knowledge of the future might be used to overthrow them. Many religious cults had hitherto used divinations (visions to foretell the future), but this practice was discouraged. Ironically, the emperors most active in the suppression of the hallucinists were those whom history has judged to be mentally ill, Tiberius, Caligula, and Nero (Moss, 1963). Following the sack of Rome by the Vandals (455 A.D.), a new force, the church, assumed a dominant role in society (Kroll, 1973).

Throughout the Middle Ages (450-1450 A.D.) Christianity was the single most important influence upon the lives of millions of people. Two principle theological schools of thought existed at this time (the mystics and the scholastics) whose attitudes towards hallucinatory experiences were quite different. Those individuals whose visions were not sanctioned by the Church were accused of witchcraft (i.e., practising pre-Christian religions) or heresy and punished accordingly (Sarbin & Juhasz, 1967).

The mystics, when confronted with a "clinical" case of hallucinations were concerned with the level of the experience. That is, its degree of distance from the world of tangible reality. Using the levels of analysis of Saint Augustine (354-430 A.D.) one could "see" with the eyes, corporeally, one could "see" through the spirit, imaginatively, or one could "see" intellectually, appreciating the transcendent essence of a universal concept. Each level of "seeing" was more detached from the world and hence more desirable. Stigma was not attached to the hallucinator (MacDermot, 1971).

By contrast, the scholastics, epitomized by Saint Thomas Aquinas (1225-1274 A.D.), perceived a fundamental dichotomy between ordinary perceptions and visions. This was a split between matters of the flesh and of the soul.

Thus scholastics were primarily concerned with the source and the content of the hallucination. This necessitated a church approved examination, since an event which bridged the gap between body and soul was of major importance. The examiner had to determine if the visions were due to material causes, the influence of Satan, or from God (Sarbin & Juhasz, 1967). The church had a vested interest in examining those individuals who hallucinated, since its role was to save them from the influence of Satan. Mental illness, as manifested by hallucinations, was often viewed as a moral sin, that is, a giving in to temptation. The individual was seen as personally responsible for this and subject to punishment by the Inquisition (Johnson, 1978).

1.3.2 Hallucinations and Mental Illness

With the coming of the Renaissance (1450-1600 A.D.) some church writers proposed the idea that certain visions might be due to illness, rather than possession or moral weakness. The individual so afflicted was therefore not personally responsible for their hallucinations. As a result of the differentiation of visions due to illness from those due to demonic possession, came an increased role for physicians. Consequently medicine, once more, became involved in the assessment and treatment of hallucinists (Kroll, 1973; Sarbin, 1967).

The idea that hallucinations could be a sign of physical illness took considerable time to develop, demonology and witchcraft charges still led to active persecution in the early 17th century (Neugebauer, 1979). It was not until the late eighteen and early nineteenth centuries that the concept of hallucinations was formalized by such writers as Erasmus Darwin (1796), Esquirol (1817), Abercrombie (1830), and de Boismont (1845), (cited in Hunter and MacAlpine, 1963, pp. 547, 731, 802, 1058). As a sign of insanity, hallucinations acquired the negative connotation that still persists to the present day. In the provocative study by Rosenhan (1973), eight people were hospitalized in mental institutions based solely on the presence of auditory hallucinations consisting of a single word.

1.3.3 Normal Hallucinations

The current association between hallucinations and mental illness began in the Renaissance and is still present today. However, not all hallucinations can be viewed as signs of psychopathology. Normal people can and do experience hallucinations. Surveying 488 societies, Bourguignon (1970) found that 437 (89%) had some form of ritualized hallucinatory states. Additionally, individuals were taught how to hallucinate and what to expect in their hallucinations. A survey of normal subjects conducted by the English Society for Psychical Research and first published in 1894 asked:

> Have you ever, when believing yourself to be completely awake, had a vivid impression of seeing or being touched by a living being or inanimate object,

> or of hearing a voice; which impression, so far as you could discover, was not due to any external physical cause? (Parish, 1897, p. 83).

Of the 27,392 replies received from France, America, Germany, and England, 3,271 individuals (12%) indicated that they had experienced hallucinations. More women than men reported having had hallucinations (14.6% females, 9.8% males). A more recent study by Posey and Losch (1983-84) surveyed the frequency of auditory hallucinations among college students. Of 375 subjects, 265 (71%) reported brief auditory hallucinations while awake. Mott, Small, and Anderson (1965) in their comparative study of 50 schizophrenics, 50 alcoholics, and 50 normals report hallucinatory frequencies of 76, 86, and 34 percent, respectively, across all sensory modalities.

Writers with an anthropological perspective frequently point out the positive aspects of hallucinations by looking at the function of the hallucination for the individual in his unique cultural milieu. Hallucinations can function as a means of sensory self-stimulation that maintains interest in a barren environment (Al-Issa, 1977), as a means of personal problem solving (Bauer, 1970; Sarbin & Juhasz, 1967), as a form of stress reduction (Weinstein, 1962), as a source of knowledge in primitive cultures (Bourguignon, 1970), as a pleasurable experience (Bleuler, 1950), or as a way of obliterating individual consciousness in the search for religious ecstasy (MacDermot, 1971). This does not diminish the association between hallucinations and mental illness, but rather serves to broaden the concept of hallucinations. In and of themselves, hallucinations need not always be manifestations of psychopathology (Forrer, 1960). But they can be, and in the western world they are usually associated with mental illness (Johnson, 1978; Linn, 1977). In addition, the variety of different etiologies of hallucinations also suggests that they should not be solely linked to mental illness.

1.4 Causes and Methods of Assessing Hallucinations

Baldwin (1970) cites the following as causes of hallucinations: sensory deprivation, toxic states, infections, organic lesions, epilepsy, migraines, alterations of body temperature, insanity, starvation, sensory monotony, and psychoactive drugs. While this list is not exhaustive, it does serve to illustrate the range of states associated with hallucinations and that they all affect the functioning of the brain.

Similarly, a variety of different methods have been used to assess hallucinations. These include verbal self-report (Siegel, 1977; Siegel & West, 1975), questionnaires (Bell, Billington, & Becker, 1985; Bentall & Slade, 1985a; Jakes & Hemsley, 1987; Launay & Slade, 1981; Posey & Losch, 1983; Young, Bentall, Slade, & Dewey, 1985, 1986), EEG measures (Heath & Walker, 1985; Koukkou & Lehmann, 1976; Stevens & Livermore, 1982), psychophysiological assessment (Inouye & Shimizu, 1970; Juninger & Rouscher, 1987, McGuigan, 1966), recording actual hallucinated speech (Green & Preston, 1981; Gould 1948, 1949, 1950), regional cerebral blood flow (Kurachi et al., 1985; Uchino et al., 1984), and CT scanning (Ota et al., 1986; Takahashi et al., 1981; Uchino et al., 1986).

1.5 Neuropsychological Investigations of Hallucinations

Although many different and varied methods of assessments of hallucinations have been employed, there is a significant dearth of neuropsychological investigations into this area. An extensive search of the literature has revealed only one published study specifically making use of neuropsychological measures to investigate hallucinations. This was a study by McKay, Golden, and Scott (1981) who utilized the Luria-Nebraska Neuropsychological Test Battery (Golden, Hammeke, & Purisch, 1980) to examine auditory and visual hallucinations in a sample of 40 hospitalized, assaultive, psychiatric patients with a substantial drug history. They found evidence of a general cognitive impairment with a left frontal focus in the auditorily hallucinated group (N=10). The group with visual hallucinations (N=10) showed no evidence of neuropsychological impairment.

These authors point out that left frontal lobe damage alone is not sufficient to produce auditory hallucinations. These subjects were also impaired on measures of right temporal and left parietal-occipital function. They also note that the normal test profiles produced by the visual hallucinators could have been due to resolution of a temporary neurophysiological toxicity resulting from drug or alcohol abuse. While the deficits of the auditorily hallucinated group were more consistent with chronic and severe cognitive and neurological dysfunction. Subjects were grouped according to predominant type of hallucination and were heterogeneous as to diagnosis and drug/alcohol use. Diagnostic criteria are not reported, although schizophrenia was the most common diagnosis in the study. Analysis of variance showed that the auditory, visual, and non-hallucinating groups (N=20) did not differ significantly on age, education, chronicity, or current psychotropic medication. The authors note that their study was limited by its small sample size, which precluded the use of multivariate statistics, and the fact that all subjects had a history of assaultive behavior. They also state that such individuals are likely to have neurological impairment resulting from their behaviors, thereby confounding the effects of an independent hallucinatory mechanism.

1.6 Neuropsychological Tests as Measures of Cerebral Functioning

As a further exploration of the neural consequences of hallucinations this investigation will utilize a modified Halstead-Reitan Neuropsychological test battery.

The reliability and validity of neuropsychological tests to assess the integrity of cerebral functioning has been extensively documented (Boll, 1981; Chapman & Wolff, 1959; Filskov & Goldstein, 1974; Halstead, 1947; Hevern, 1980; Klesges, Fisher, Pheley, Boschee, & Vasey, 1984; Klove, 1974; Lezak, 1983; Matarazzo, Matarazzo, Weins, & Galo, 1976; Reitan, 1955, 1966; Reitan & Davison, 1974; Reitan & Wolfson, 1985; Rubino, 1970; Russell, 1983; Schneider, Goldman, Kleinman, Goldfader, & Snow, 1976; Snow, 1981; Stuss & Trites, 1977; Swiercinsky & Leigh, 1979; Tsushima & Wedding, 1979; Vega & Parsons, 1967; Weins & Matarazzo, 1977).

In addition, a substantial body of literature exists that has utilized neuropsychological measures to investigate brain dysfunction in psychiatric syndromes (Flor-Henry, 1983a; Flor-Henry, Fromm-Auch, Tapper, & Schopflocher, 1981; Golden, 1977; Golden, Moses, Coffman, Miller, & Strider, 1983; Goldstein, 1978, 1984; Goldstein & Shelly, 1972; Grant & Adams, 1986; Heaton, Baade, & Johnson, 1976; Heaton & Pendleton, 1981; Insel, Donnelly, Laleka, Alterman, & Murphy, 1983; Malloy, Small, Miller, Milstein, & Stout, 1982; Marin & Tucker, 1981; McAllister, 1981; Miller, 1985; Otto, Yeo, & Dougher, 1987; Rutter, 1983; Seidman, 1983; Strider, Chung-Chu, Golden, & Bishop, 1985; Taylor, Redfield, & Abrams, 1981; Wexler, 1980; Yeudall, 1977; Yeudall, Fedora, & Fromm, 1987).

More recently, neuropsychological investigations of schizophrenia have begun to focus on specific symptoms of this disorder (Bilder, Mukherjee, Rieber, & Pandurangi, 1985; Green & Walker, 1985; Langell,. Purisch, & Golden, 1987; Silverstein & Arzt, 1985; Silverstein & Zerwic, 1985). This approach, emphasizing symptoms rather than diagnosis, has been strongly advocated by Persons (1986) who argues that the study of specific psychological phenomena avoids many of the problems inherent in delineating and classifying diagnostic groups.

1.7 Definitions of Hallucinations

Before discussing the specific purpose of the dissertation, it is important to briefly review definitional and classification issues as they pertain to hallucinations. While it is common knowledge that people who hallucinate "see" things that are not there, or "hear" voices no one else can hear, it is much more difficult to rigorously specify what exactly constitutes a hallucination. Sully (1881) suggested that hallucinations involved "... the projection of a mental image outwards when there is no external agency answering to it" (p. 113). Prior to this in 1817 the French psychiatrist Esquirol, cited in Hunter and MacAlpine (1963, p. 733) asserted that "A person is said to labor under a hallucination, or to be a visionary, who has a thorough conviction of the perception of a sensation, when no external object, suited to excite this sensation, has impressed the senses." Following the translation of two of his papers by Liddell in 1833 as, Observations on the illusions of the insane, and on the medico-legal question of their confinement, Esquirol elaborated on his definition of hallucinations:

> In hallucinations everything happens in the brain (mind). The visionaries, the ecstatics are people who suffer from hallucinations, dreamers while they are awake. The activity of the brain is so energetic that the visionary, the person hallucinating ascribes a body and an actuality to images that the memory recalls without the intervention of the senses.

Earlier, Crichton (1798) cited in Parish (1897, p. 13) differentiated between hallucinations and illusions as "... errors of the mind by which in the one case ideas are taken for matters of fact, and in the other case real objects are falsely represented, but without any general disturbance of the intellectual faculties." More recently Woodworth and Marquis (1947) suggested that "a hallucination is a memory image taken for a perception; it is something recalled or built up out of past experience and taken for a present objective fact" (p.565).

Brain (1969) defined hallucinations "... as mental impressions of sensory vividness occurring without external stimulus, but appearing to be located, or to possess a cause located, outside the subject" (p. 980). Writing from an anthropological perspective, Weinstein (1962) argued that "... a hallucination is a highly condensed symbol that has the quality of reality because it is an element in a pattern of social relatedness" (p. 147). More recently, Hinsie and Campbell (1970) defined hallucinations as "An apparent perception of an external object when no such object is present" (p. 333). Finally, a comprehensive definition offered by Siegel (1977) states that a hallucination is "a false sensory perception in the absence of an actual external stimulus. May be induced by emotional and other factors such as drugs, alcohol, and stress. May occur in any of the senses" (p. 132).

1.7.1 Summary of Characteristics of Hallucinations

From the above definitions, several elements can be isolated. Firstly, is the idea of false sensory impressions; the hallucinist is deceived, he or she believes their perceptions to be veridical, but they are not. Second, since hallucinations cannot be observed directly (except by the hallucinist) their presence must be inferred or deduced from the verbalizations and behaviors of those people who are experiencing hallucinations (Sarbin, 1967). This necessitates the presence of "experts," qualified to assess the extent, significance, quality, and function of hallucinations (i.e., psychiatrists, psychologists, social workers, etc.). In fact, one way to trace the development of the conceptualization of hallucinations is by investigating which societal agencies were informed with hallucinators.

A third point is that the content of the hallucination is ultimately derived from the memory and imaginal processes of the hallucinator. This suggests that the content of the hallucination has dynamic relevance to the hallucinist, in other words the phenomenology of a hallucination is meaningful and not random. This does not imply causality, but rather function; one may determine the "meaning" of a specific hallucination in terms of a particular individual without saying anything about how that event took place (Barnett, 1965). This is an ideal principle and it may not be possible to symbolically analyze all hallucinations encountered in clinical practice, e.g. the toxic confusion of delirium tremens. Even here some success has been achieved, see Bauer (1970) for a case-study account relating alcohol hallucinosis to feelings of ambivalence toward a daughter.

Fourth, in view of the large number of circumstances that can induce hallucinations (brain disease, sensory deprivation, drug abuse, dreaming, psychoses, etc.) and the variety of hallucinatory experiences possible, they are best regarded as multi-modal and multi-casual in origin. The situation is analogous to Psychology's attempt to find a single test of "brain damage" by treating it as a unitary concept. One must recognize that in brain damage, as in hallucinations, different etiologies may produce a similar phenomenology (Evarts, 1962).

To summarize, the definitions of hallucinations presented above suggest that the hallucinator's senses are deceived, but that he or she behaves "as if" their perceptions are veridical. Because they cannot be directly experienced by others, hallucinations are inferred events. The content of hallucinations derive ultimately from the memory and imaginal processes of the hallucinator and hence are meaningful. In addition, hallucinations are regarded as multi-modal and multi-casual events. While not clearly emerging from the definitions above, an important dimension of hallucinatory experience is the spontaneous nature and lack of control individuals experience over their hallucinations. Hallucinations are typically intrusive experiences that occur without conscious control by the individual (Slade, 1976c).

1.7.2 Clinical Assessment of Hallucinations

Although this investigation is not directly concerned with the clinical assessment of hallucinations, it is relevant to briefly discuss the variables involved in generating clinical information about hallucinations. Typically, a distinction is made between true and pseudo-hallucinations. In North America, the latter are hallucinations with insight, that is hallucinatory experiences which are recognized by the individual as not being true perceptions. There is some controversy over the value of this distinction for diagnosis and prognosis (Hare, 1973; Stevenson, 1983; Taylor, 1981). Early in the assessment, the clinician will make an initial judgement of whether the hallucination is due to toxic or functional causes. If the subject's sensorium is clear and he or she is not under the influence of a toxin such as drugs or alcohol, it is likely that the hallucinations are related to some form of mental illness, that is to say, they are functional in nature. Traditionally, visual hallucinations are more related to toxic or organic states, while auditory hallucinations are more common in the functional psychoses (West, 1975).

In order to determine how pervasive an individual's hallucinations are, the clinician will usually gather data about their frequency of occurrence and the amount of time that they occupy. The individual's past history of hallucinating is also relevant, when they first occurred, when they fluctuate in frequency and intensity, and when they are stable. Also the modalities hallucinated in should be investigated and their relative contribution noted. The antecedent and surrounding conditions of the hallucinations are important to know. It has been reported for certain hallucinated schizophrenics that reducing their anxiety to social situations decreases the frequency of their hallucinations (Al-Issa, 1976a). Thus the subject's mood, energy level, and social interactions at the time of the hallucinations is important.

An accurate description of the actual content of the hallucination itself is useful. This will help the clinician evaluate the specific and personal relevance of the hallucination for the individual. The clinician will also evaluate to what extent the hallucinator is behaviorally controlled by their hallucinations and in what manner the individual reacts to their hallucinations. To summarize the preceding, when clinically interviewing a hallucinated client, it is useful to evaluate the veridicality and nature of the hallucinations, the extent of the hallucinations, their antecedent and precipitating conditions, the phenomenology, and the behavioral responses to (or consequences of) the hallucinations.

1.7.3 Modifiers of Hallucinatory Experience

The previous section described the kind of information that a clinician is likely to try and elicit from a hallucinated subject. The following is a description of variables that can potentially modify the actual hallucinatory experience. In the analysis of hallucinations it has long been recognized that the personality of the hallucinator contributes uniquely to their hallucinations (Modell, 1960). In 1830, Abercrombie, writing on the topic of insanity, described how "propensities of character," ordinarily controlled, could become manifest in hallucinations.

> Thus a man of an aspiring ambitious character may imagine himself a king or a great personage; while, in a man of timid, suspicious disposition, the mind may fix upon some supposed injury, or loss either of property of reputation (cited in Hunter & MacAlpine, 1963, p. 802).

Abercrombie then went on to suggest that old memories could be found represented within hallucinations and that delusional misinterpretations of somatic sensations could lead to hallucinations. Thus over one hundred and fifty years ago it was recognized that the premorbid personality of the hallucinist could affect the phenomenology of his or her hallucinations.

Also significant are the expectations that the individual brings to their hallucinatory experiences. In the western world, hallucinations are viewed as being symptomatic of psychopathology (Linn, 1977), while in more primitive societies, hallucinations are woven into the fabric of the culture. As cited previously, the anthropological survey work of Bourguignon (1970) showed 437 out of 488 societies to have some form of ritualized hallucinatory states. More importantly, individuals were taught how to hallucinate in their societies and what to expect during their hallucinations. The methods of teaching were rigorous, usually involving self-punishment, special exercises, drugs and/or deprivation of food, sleep, and sensory contact. Hallucinatory experiences were actively sought in order to establish a connection between the individual and a spirit guardian. The content of hallucinations thus induced tended to be traditional, coincident with the expectations of the hallucinator, rather than idiosyncratic as is frequently found in western culture (Wallace, 1959).

Just as an individual's expectations may modify hallucinatory experiences, so too may cultural values. Culture provides an attitudinal framework within which the individual exists. Some non-western cultures tend to be supportive of the hallucinatory experience, emphasizing its value as a source of knowledge for both the individual and his community (Bourguignon, 1970). Weinstein (1962) studying psychiatric patients in the American Virgin Islands found that children commonly figured as content in delusions and hallucinations. In this culture children were highly valued within all the different social classes and Weinstein felt that their frequent inclusion in the fantasies of psychiatric patients reflected this fact. He argued that because hallucinations occur within a social matrix, it is not unreasonable to expect that their content will reflect prominent values within the culture. A similar kind of analogy may be drawn from North American culture with its emphasis on technology. As we have become more sophisticated, the voices heard by schizophrenics which were originally explained as being transmitted by radio waves, then by TV or micro-waves, are more recently being transmitted by laser beams.

Another major difference between Western and non-Western attitudes towards hallucinators is the source of the hallucination. For non-Western societies it does not matter if the hallucination is drug induced, occurs in a setting of clear consciousness or delirium, is an especially vivid dream, comes from fatigue, hunger, or results from physical pain, it is still a valued communication (Alexander & Selesnick, 1966; Radkin, 1970). Whereas in the Western world, considerable emphasis is placed on the cause of the hallucination; had the individual just awakened or just fallen asleep, had they been drinking heavily and recently stopped, were they high on drugs, toxic on medications, or having convulsions. While these are important dimensions of the hallucinatory experience, they should not blind the clinician to the significance of the phenomenology of the hallucination (Al-Issa, 1976b).

Erasmus Darwin (1731-1802), the grandfather of Charles Darwin, recommended investigation of the content of delusions and hallucinations in order to gain psychological insights into the minds of the mentally ill. He describes several cases of delusions and hallucinations in order to:

> ..shew [sic] the utility of endeavoring to investigate the maniacal idea, or hallucination; as it may not only acquaint us with the probable designs of the patient, from whence may be deduced the necessity of confinement; but also may some time lead to the most effectual plan of cure. (cited in Hunter & MacAlpine, 1963, p.550).

As Hunter and MacAlpine observed, from reasoning such as this, one can see the attitudes that led to the beginnings of what became psychopathology and psychotherapeutic interventions.

As mentioned in the discussion of the definition of hallucinations, they are not directly observable events and their presence is inferred from the interaction of the hallucinator (patient) and his or her examiner (clinician). Typically for the hallucinist, their experiences are "real" (Straus, 1962), but it is the examiner who determines the value or significance of the hallucinations. It is the examiner's "socially approved" reality criterion that prevails (Sarbin, 1967). People who hallucinate tend to be judged negatively and it has been strongly argued by Sarbin and Juhasz (1967) that the term hallucination, in Western culture, is a pejorative term which is used to imply that the person is insane. Recently, Stevenson (1983) suggested that in view of the large number of non-mentally ill people who experience hallucinations, unshared sensory experience in normals be called "idiophany" (from the Greek idios meaning private, and phainomai meaning to appear) and that the term hallucination be reserved for unshared sensory experience in the mentally ill.

In summary, several factors that modify hallucinatory experience have been presented. These include, the premorbid personality characteristics of the hallucinator, the individual and cultural expectations within which the hallucination occurs, the perceived cause of the hallucinations, the phenomenology of the hallucinations, and the interaction of the examiner and the hallucinist. Writers with a traditional medical orientation are more concerned with etiological factors, while those whose persuasion is more psychological or anthropological look towards the function of the hallucination for the individual and their culture.

1.8 Classification of Hallucinations

1.8.1 Drug Induced Hallucinations

In terms of how hallucinations are classified in practice, no specific system has been generally accepted. The way in which hallucinatory phenomena are grouped often reflects the focus of the investigator. Thus, Siegel (1977) studying the effects of hallucinogenic drugs, trained his volunteers to record their effects on an "experiential typewriter" which allowed them to keep up with the flow of visual images produced by drugs. In contrast, Malitz, Wilkens, and Esecover (1962) studied the adaptational value of LSD induced hallucinations in both normal subjects and chronic schizophrenics. While these authors were studying the commonality between a "model" drug induced psychosis in normals compared to the psychosis of schizophrenia, it is interesting to note that the visual forms described by subjects in this study were strikingly similar to those reported by Siegel's (1978) trained observers. These geometrically shaped visual hallucinations were first systematically described as effects of mescaline intoxication by Kluver (1926) at the University of Chicago.

1.8.2 Psychiatric Hallucinations

Within the field of psychiatry the formal qualities of hallucinations have long been used as an aid to diagnosis. First-rank or Schneiderian symptoms emphasize the role of specific kinds of hallucinations in the diagnosis of schizophrenia (Schneider, 1959). Lowe (1973) developed a discriminant function to classify four diagnostic groups based on the types of hallucinations experienced by each. He concluded that, "The findings suggest that phenomenological characteristics of hallucinations may be used as discriminatory indicators for differential diagnosis among psychotics" (Lowe, 1973, p. 630). However two years previously Goodwin, Alderson, and Rosenthal (1971) had examined a group of 117 psychiatric patients selected on the basis of the symptom hallucinations, rather than diagnosis. They culled the psychiatric literature to determine clinical opinion concerning the diagnostic and prognostic value of different kinds of hallucinations. Of the nine hypotheses that they developed, none were supported. They found hallucinations to be nonspecific with regard to diagnosis. Similarly, Frieske and Wilson (1966) compared visual hallucinations in patients with schizophrenic, organic, and affective psychoses. They found hallucinations to be similar in terms of perceptual qualities across all three groups. This lack of phenomenological, diagnostic, or prognostic specificity has been found by others (cf. Derker, & Chambers, 1978; El-Tayeb, & Zarroug, 1975; Hellerstein, Frosch, & Koenigsberg, 1987; Jansson, 1968, Larkin, 1979; Lewinsohn, 1968, 1980).

1.8.3 Hallucinations in Brain Disorders

Investigators working directly with the brain in neurosurgical environments have developed methods of classification of both spontaneous and electrically elicited hallucinations. The research of Penfield and Perot (1963) on temporal lobe epileptics divided the mental events associated with epilepsy into several categories. Firstly, they could be spontaneous seizure events or they could be electrically induced. An experiential hallucination occurred during a spontaneous seizure, while an experiential response occurred as a result of electrical stimulation of temporal lobe cortex. The content of the hallucinations involved things that the patient had previously seen, heard, or experienced. A second kind of phenomena that could occur either spontaneously with a seizure or be elicited was the interpretative response or interpretative illusion. This referred to an altered interpretation of ongoing present experience and involved the patient experiencing unusual "feelings" such as deja vu or emotions such as fear or loneliness. Illusions of interpretation resulting from electrical stimulation were further subdivided into auditory, visual, emotional, or recognition illusions. Experiential hallucinations and experiential responses were classified as auditory, visual, combined auditory and visual, or unclassified. In summary, this classification scheme was applied to mental sequelae associated with temporal lobe epilepsy. Events could occur spontaneously during seizures or be elicited by electrical stimulation of the temporal cortex. Illusions involved altered interpretation of present experience, while hallucinations involved recognizing or reliving past experiences.

The idea that electrical stimulation of the temporal cortex released stored memories that were somehow "relived" was challenged by Horowitz and Adams (1970). They placed depth electrodes in the temporal lobes of patients with intractable epilepsy. Visual events were categorized as hallucinations, pseudohallucinations, elementary sensations, and perceptual distortions. Hallucinations were images occupying space external to the subject. Pseudohallucinations were images not projected externally or experienced as real perceptions. Elementary sensations were unorganized vivid images and perceptual distortions involved alteration in the experience of actual objects. The authors found that electrical stimulation of the same site produced different perceptual experiences in the same patient and that rather than representing prior experience these events could sometimes be related to the patient's pre-operative mental content.

Roberts (1984) separates auditory and visual hallucinations into formed and unformed. Unformed visual hallucinations involve spots, lines, or patterns and may be due to occipital lobe lesions, hallucinogen use, migraine, end-organ disease, optic nerve lesions, or delirium. Formed visual hallucinations involving objects, other people, or animals may be due to depressive psychosis, schizophrenia, delirium, migraine, or diffuse cerebral disease. Those formed visual hallucinations involving landscapes or other complex scenes may be caused by temporal lobe disease, midbrain and thalamic lesions, schizophrenia, affective illness, or end organ disease. Auditory hallucinations of an unformed type such as whistling, tinnitus, or clicking can be caused by lesions of the superior temporal gyrus, salicylate abuse, or disease of the middle ear and auditory nerve. Formed auditory hallucinations, that is voices or music, are found in schizophrenia, depression, alcohol hallucinosis, delirium, end organ disease, temporal lobe disease, and amphetamine psychoses.

A different scheme of classification for auditory and visual hallucinations has been utilized by Hecaen and Albert (1978). Visual hallucinations are separated into elementary and complex forms. Elementary hallucinations are essentially non-patterned flashes of light. Complex visual hallucinations are described as esthesic, associated with dreamy states, with oneirism, or with ophthalmological lesions. Esthesic hallucinations typically occupy the entire visual field, occur with a clear sensorium, remain in memory, and appear real and novel to the subject. Visual hallucinations associated with dreamy states occur in a context of modified consciousness. They are hard to remember and the experience has an unreal quality. They may involve reliving a previous experience or a distortion of present experience. Hallucinations occurring with oneirism are characterized by substantial alterations of consciousness and temporospatial disorientation. Visions are usually complex, associated with emotional distress, and the individual is amnestic for the experience. Hallucinations associated with ophthalmological lesions include the Charles Bonnet syndrome and monocular hallucinations with positive scotoma.

Auditory hallucinations are also classified as elementary and complex, with complex auditory hallucinations being subdivided into musical and verbal forms. Elementary auditory hallucinations are indistinct noises that are hard to describe and are frequently recognized as being pathological by the hallucinator. Complex musical hallucinations may be stereotyped in nature and are often very clear sensorially. They may be purely instrumental or contain both singing and music. Complex auditory hallucinations can involve single words or extended monologues. More than one voice may be heard and usually they are less clear than musical hallucinations. Affective elements are often present. Individuals experiencing complex musical or verbal hallucinations fall into two major groups. Those whose hallucinations appear externally, believe their hallucinations are real and have a clear sensorium and those whose hallucinations are characterized by feelings of strangeness and clouded consciousness.

1.8.4 Psychological Aspects of Hallucinations

From a psychological perspective, a useful classificatory approach is that of Sedman (1966) who compared the qualitative features of imagery, pseudohallucinations, and true hallucinations. This was done in states of clear consciousness, half-walking states, and clouded consciousness. Imagery was defined as an unusual mental experience that lacked the concrete reality of a perception and that appeared in inner subjective space (i.e., in the mind's-eye). Pseudohallucinations were defined as hallucinations that were perceived through the senses but recognized as non-veridical perceptions. Typically they were psychologically meaningful to the individual. True hallucinations were defined as experiences perceived through the senses and accepted as real. These, in a state of clear consciousness, were common in schizophrenia. Imagery and pseudohallucinations, in a state of clear consciousness, were related to personality characteristics, rather than psychoses. Additional phenomenological classifications of hallucinations are discussed in Aggernaes (1972), Aggernaes and Nyeborg (1972), Berrios (1985b), Jaspers (1963), and Smythies (1956). Common to these reports is an emphasis on the similarity of real perceptual experiences and true hallucinations.

1.8.5 Summary

Hallucinations are usually classified according to their form and content. The focus of the investigator determines what elements are emphasized. This will differ depending on whether drug induced hallucinations are being studied, whether the hallucination is used for psychiatric diagnosis, phenomenological investigation, or results from direct brain stimulation. Within the context of the present study the focus is on the neuropsychological correlates of the hallucinations that are found in hospitalized psychiatric patients. There is an extensive literature concerning hallucinations in normals (see Johnson, 1978) but, except for review purposes, this is largely outside the scope of the present study, which is an attempt to delineate patterns of neuropsychological test scores in hallucinated psychiatric inpatients.

1.9 Purpose of the Dissertation

Specifically, this study will investigate whether differences in cognitive abilities exist between hospitalized hallucinated and non-hallucinated psychiatric patients. That is, are there different neuropsychological test patterns between these two groups and are these test patterns different from those produced by normal control subjects? The investigation rests on the assumption that hallucinations, as experienced by the mentally ill, are a manifestation of disordered brain functioning. An attempt will be made to link the psychiatric symptom, hallucinations, with patterns of cerebral dysfunction, as evidenced on neuropsychological tests (Miller, 1985). Neuropsychological tests are, of course, assumed to be sensitive to cerebral pathology. Simply stated, the problem investigated in this study is, what are the neuropsychological correlates of hallucinations in psychiatric patients.

1.10 Synopsis of Method of the Investigation

The method of investigation used in this project is the case-control study (Schlesslman, 1982). It is a retrospective observational approach in which cases with the condition under study (hallucinations) are compared against controls who do not have the condition under study. The method is exploratory, working from effect to hypothesized cause, and it tries to identify associated variables with the condition under study. Groups are compared for differences and the information generated may be used to develop hypotheses testable by experimental manipulation. The advantage of the case-control approach is that it allows for the investigation of multiple hypotheses in situations where only limited information is available concerning the phenomena under study. It is the logical precursor to analytic studies with precisely defined hypotheses. The function of this retrospective approach is not to determine causal relationships, but to demonstrate associations between relevant variables that, once isolated, can be more precisely investigated (Bailar, Louis, Lavori, & Polansky, 1986; Schlesslman, 1982).

The following questions will be addressed:

1) Are hallucinated psychiatric patients different from normal controls, as assessed by a battery of neuropsychological tests?
2) Are hallucinated psychiatric patients different from non-hallucinated psychiatric patients, as assessed by a battery of neuropsychological tests?
3) Are unselected hallucinated psychiatric patients different from selected hallucinated psychiatric patients, as assessed by a battery of neuropsychological tests?

The term "selected patients" refers to those subjects diagnosed according to specific research criteria (Taylor & Abrams, 1978), while "unselected patients" are those who have been assessed/diagnosed routinely as part of the normal flow of hospital admissions. For the selected and unselected patient groups both hallucinated and non-hallucinated cases are available.

1.10.1 Subject Selection and Method of Analysis

The selected sample consists of 54 hospitalized psychiatric patients who satisfy the diagnostic criteria of Taylor and Abrams (1978) for Schizophrenia, Mania, and Endogenous Depression. The unselected sample consists of 107 hospitalized psychiatric patients who are representative of psychiatric patients at Alberta Hospital Edmonton. These are all hallucinated, non-forensic patients who have been formally presented at diagnostic case conferences. Control data consist of 182 normal volunteers with a negative psychiatric and neurological history as determined by interview (Fromm-Auch & Yeudall, 1983; Yeudall, Fromm, Reddon, & Stefanyk, 1986; Yeudall, Reddon, Gill, & Stefanyk, 1987).

Analysis of variance procedures were used to compare hallucinated subjects with normals, hallucinated subjects with non-hallucinated subjects, and selected hallucinators with unselected hallucinated subjects. Comparisons with normal controls were stratified by age and sex on data from fifteen neuropsychological tests and the Wechsler Adult Intelligence Scales. In order to establish the presence of hallucinations an extensive chart review was conducted for the selected sample. This involved checking the admission notes, full history, nursing notes and, where available, the discharge summary. For the unselected sample of hallucinators, the presence of hallucinations was established by history, case conference, patient interview, or a combination of the above.

1.11 Scope of the Dissertation

It is the aim of this investigation to use cognitive, motor, and sensory-perceptual tests to assess hallucinated psychiatric patients in an effort to derive a model of the neuropsychological correlates of hallucinations. Within the psychiatric population, hallucinations are regarded as a manifestation of disordered brain functioning. Neuropsychological tests have been chosen because of their documented sensitivity to disordered cerebral processing. To review all of the literature on the topic of hallucinations is not possible and consequently certain areas have been omitted. These include hallucinations in normal subjects (see Johnson, 1978; Posey & Losch, 1983-84; Spanos & Henderikus, 1979) and hallucinations in childhood (Aug & Ables, 1971; Egdell, & Kolvin, 1972; Garralda 1984a, 1984b, 1984c; Kemph, 1987; Rothstein, 1981; Schneier & Libow, 1986). The literature on drug induced hallucinations is beyond the scope of this investigation (for an introduction to different aspects of this problem see Brawley & Duffield, 1972; Cohen, 1984; Miller, 1985; West, 1968). Delusions also fall outside the range of this study, although it is recognized that they are closely associated with hallucinations (Cummings, 1985).

1.12 Plan of the Dissertation

Following is an outline of the investigation, which will be divided into six chapters. The first chapter was a general introduction to the problem of hallucinations and examined what constituted a hallucination. The evolution of the concept of hallucinations and the use of neuropsychological tests to assess brain function was discussed. Subjects and method were briefly described. The review of the literature falls into chapters II and III. Chapter II is a discussion of organic causes of hallucinations and the behavioral and pharmacological strategies used to treat hallucinations and Chapter III is a description of neuropsychological, cognitive and speech dysregulation models of hallucinations. Chapter IV is the design section of the dissertation, including subject description, assessment variables, procedures used, and methods of data analysis. Chapter V describes the results of the investigation and Chapter VI is a general discussion which describes neuropsychological sequelae of hallucinations and proposes future lines of inquiry into the problem of hallucinations.

Chapter II

Review of the Literature: Part I, Organic Causes of Hallucinations, Behavioral and Pharmacological Strategies of Treatment

2.1 Introduction

This chapter examines the relationship of organic brain damage to hallucinations. Thus only studies using subjects with documented brain damage are included. The purpose of the chapter is to review where in the brain different kinds of hallucinations are elicited, not how the hallucinations are produced. This is discussed in a separate chapter specifically reviewing theories of hallucinations. For ease of presentation, the material is organized around hallucinatory modality, rather than site of lesion. Gustatory, olfactory, tactile, auditory, and visual hallucinations are discussed.

2.2 Gustatory Hallucinations

The literature on gustatory hallucinations is quite sparse. This is, in part, due to the grouping of gustatory and olfactory hallucinations together, as well as their rare occurrence. Hausser-Hauw and Bancaud (1987) reviewed the literature on frequency of gustatory hallucinations and found a range of .09% to 29.1% in samples of epileptic patients. Sample sizes ranged from 35 to 1,102 cases. The frequency of gustatory hallucinations depended on whether samples dealt with mixed forms of epilepsy, psychomotor epilepsy, uncinate fits, or temporal lobe epilepsy. The authors report that 4% of their sample of 718 epileptic patients experienced gustatory hallucinations as part of their seizure.

Typically, gustatory hallucinations involve an unpleasant taste that may be described as salty, bitter, like bile, sweet, or like acetone. They may precede or be a part of an epileptic seizure. Traditionally the temporal lobes have been viewed as the site of origin of gustatory hallucinations (Pincus & Tucker, 1985; Roberts, 1984). In fact, the uncinate fit, which consists of oral-facial movements, olfactory and gustatory hallucinations, and feelings of fear is regarded as highly suggestive of a temporal lobe lesion (Angevine & Cotman, 1981; Critchley & Rossall, 1978).

Hausser-Hauw and Bancaud (1987) in their study of 30 epileptic patients with gustatory hallucinations suggest that the parietal operculum and/or Rolandic operculum are the critical sites of origin for gustatory hallucinations. Operculum is the name given to the portions of the frontal, temporal, and parietal lobes adjacent to the central sulcus that overlie the insular cortex (Carpenter, 1973). Their subjects were investigated by means of stereoelectroencephalographic exploration which involved intracerebral depth electrode placement. This allowed for precise correlation between anatomic site of stimulation and real-time behavioral sequelae (i.e., seizure activity). The authors based their conclusions on the following findings. First of all, gustatory hallucinations were predominant with parietal lobe seizures and with stimulation of the parietal operculum and/or rolandic operculum, while with temporal lobe seizures they were one symptom among many. Secondly, a gustatory hallucination was induced in a case of temporal lobe epilepsy by stimulation of the parietal operculum. The individual had never complained of gustatory hallucinations before. Whereas, in epileptics already experiencing this phenomenon, stimulation of the parietal and rolandic operculum evoked gustatory hallucinations. Thirdly, temporal lobectomy did not abolish gustatory hallucinations in three of their cases. Thus while gustatory hallucinations can be manifested in parietal, temporal or temporo-parietal seizures, Hausser-Hauw and Bancaud concluded that the parietal and/or rolandic operculum are the crucial sites of disorganization.

2.3 Olfactory Hallucinations

As with gustatory hallucinations, olfactory hallucinations are not a frequent event, except within the temporal lobe epilepsies (Asaad & Shapiro, 1986; Roberts, 1984). In addition, they are also found within the schizophrenias, where part of the clinical problem is to distinguish the delusional belief of an offensive body odor from an hallucination of offensive body odor (Cavenar & Brodie, 1983). Hallucinated odors are typically offensive, though not exclusively so (Rubert, Hollender, &

Mehrhof, 1961). An extensive examination of olfactory hallucinations by Pryse-Phillips (1971) concluded that, as a symptom, they are much more common than is generally believed. He describes an olfactory reference syndrome, similar to depressive illness which may also manifest olfactory hallucinations. The 11 temporal lobe epileptics in this study experienced their hallucinations as extrinsic to their bodies and perceived the hallucinations to be part of their epileptic illness.

A search of the literature did not elicit articles concerning the specific pathophysiology of olfactory hallucinations. Nevertheless, the neuroanatomic pathways of smell have been traced (Angevine & Cotman, 1981) and olfactory hallucinations, in temporal lobe epilepsy, are associated with irritative lesions of the parahippocampal gyrus, uncus and areas surrounding the amygdaloid nuclear complex (Carpenter, 1973; Matzke & Foltze, 1972).

2.4 Tactile Hallucinations

Hallucinations of touch are associated with multiple etiologies. They may occur in psychosis associated with mental illness, in organic brain syndromes such as dementia, in toxic brain states (e.g. delirium tremens, cocaine, and amphetamine intoxication) or as part of the syndrome of delusional parasitosis (Berrios, 1985a). In addition, Takeda et al. (1985) offer a neuroendocrinological formulation for a case of tactile hallucinations. Specific cerebral mechanisms of tactile hallucinations have not, as yet, been identified, and in view of the diversity of etiologies linked to this condition, it is unlikely that they will be in the near future. Berrios (1982) in a review of the etiological and conceptual issues surrounding tactile hallucinations, cites cases resulting from brain injuries, dementia, diabetes, and hypophyseal tumor.

Haptic hallucinations are frequently found with chronic cocaine intoxication, typically as a creeping sensation under the skin. This formication is most common after prolonged intravenous use of the drug (Asaad & Shapiro, 1986; Roberts, 1984). Siegel (1978) proposed that the hallucinations of cocaine abuse were related to seizure-type discharges within the temporal lobes and increased activity of the reticular activating system.

The syndrome of delusional parasitosis, in which individuals believe that they are infested with lice, vermin, or small animals that they feel beneath the skin, has been linked to a variety of organic disorders involving the whole body (Berrios, 1985a, 1985b). The work of Takeda et al. (1985) suggests a role for the neurotransmitter dopamine in this syndrome. A case of delusional parasitosis secondary to a hypophyseal tumor that also resulted in high serum prolactin levels was treated with a sequential combination of bromocriptine and haloperidol. These drugs act on the tuberoinfundibular and mesocortical dopamine pathways. The authors hypothesized that the bromocriptine had inhibited prolactin secretion by the hypophyseal tumor. This in turn reduced tuberoinfundibular dopamine levels, at which point haloperidol became effective as a dopamine antagonist in the mesocortical pathway and produced an antipsychotic effect. Autopsy findings showed no basilar artery compression or cortical degeneration resulting from the tumor. Psychiatrically, the patient had the delusional belief that ticks were crawling all over her head and back, otherwise her mental status was normal.

In summary, little is known about specific cerebral mechanisms to account for tactile hallucinations. While common in cases of chronic cocaine or amphetamine abuse, they are infrequently seen in psychiatric or neurological populations (Asaad & Shapiro, 1986; Walker & Cavenar, 1983). When found as a monosymptomatic delusion, organic causes should be investigated (Berrios, 1985a).

2.5 Auditory Hallucinations

Complex auditory hallucinations in a setting of clear consciousness, are intimately associated with schizophrenia (Asaad & Shapiro, 1986; Roberts, 1984; Walker & Cavenar, 1983). However, not all auditory hallucinations are collaterals of mental illness. Those resulting from cortical lesions are typically manifestations of epilepsy (Hecaen & Albert, 1978). These authors, summarizing several reported series of cases in the literature, cite frequency of occurrence figures of 1%

to 18%. They also note that in a series of cases investigated by the senior author, where lesion laterality data was available, the left hemisphere was implicated in 13 out of the 16 cases with auditory hallucinations. The role of temporal cortex in hallucinations is discussed in an editorial by Slade (1976c) who notes that in the classic stimulation study of Penfield and Perot (1963) meaningful auditory and visual hallucinations occurred only with temporal lobe stimulation.

These authors reviewed all temporal lobe surgery cases from the Montreal Neurological Institute since it was founded in 1934. Of 520 patients, 40 (7.7%) manifested experiential hallucinations upon stimulation. Experiential hallucinations involved reliving events previously seen or heard or experienced by the patients. Typically, they were recognized by the patient as coming from their past. Fifty-three patients (10%) had experiential hallucinations as part of their epileptic attacks. In summarizing their data, Penfield and Perot (1963) reported that for stimulation induced hallucinations, there was a trend for the right temporal lobe to produce more auditory and visual responses than the left. For patients with hallucinations as part of their ictal pattern, visual hallucinations were clearly a right temporal phenomena. This finding was consistent with their review of the literature. Auditory hallucinations were more frequent in the left temporal lobe, but the difference was too small to have any lateralizing value. The areas most likely to result in hallucinatory activity when stimulated were the lateral and superior surfaces of the first temporal convolution. Interestingly, stimulation of Heschl's gyrus (Brodmann's area 41), the primary auditory cortex in man, did not result in any experiential responses.

Two additional studies of epileptic patients have also failed to show clearly lateralized effects for hallucinations. An investigation by Toone, Dawson, and Driver (1982) examined the relationship between psychosis and epilepsy using computerized axial tomography. They found an absence of right sided lesions in their index group of 57 patients, of whom 22 were hallucinated. Tucker, Price, Johnson, and McAllister (1986) examined the phenomenology of 20 cases of complex partial seizures. Auditory hallucinations were present in 10 cases and visual hallucinations in 8 cases. Of the 19 patients with abnormal EEG findings, 13 showed temporal lobe dysfunction. Most of these were bilaterally abnormal, with the remainder showing a left sided emphasis.

Using a signal threshold technique Bazhin, Wasserman, and Tonkonogh (1975) found the results of 30 hallucinated paranoid schizophrenics to be similar to those of subjects with left temporal lobe pathology. These patients demonstrated an asymmetric pattern of detection thresholds characterized by an increase in the right ear threshold. This effect became more pronounced with increased task difficulty. Additional evidence of cortical involvement in auditory hallucinations comes from the findings of a group of Japanese investigators. Takahashi et al. (1981) compiled comprehensive CT scan data on 280 schizophrenics, diagnosed according to the criteria of the International Pilot Study of Schizophrenia (1973) and subtyped according to the International Classification of Diseases - 9th revision of the World Health Organization (1977). While the focus of the study was on structural brain damage in schizophrenia, the authors noted that hallucinations were associated only with left temporal atrophy.

In addition to the association of hallucinations with cortical brain damage, the neurological literature provides evidence for the role of subcortical brain systems in auditory hallucinations. Cascino and Adams (1986) reported three cases of auditory hallucinations as sequelae to lesions of the tegmentum of the pons and lower midbrain. The hallucinations were of a simple, unformed type that sounded like buzzing, clanging, or bells chiming. In 2 of the 3 patients mental status was normal, while the third patient was drowsy and disoriented. Identification of the lesions was determined by clinical, CT scan, and auditory evoked response data. The following year Lanska, Lanska, and Mendez (1987) reported an additional case of elementary auditory hallucinations following a left lower pontine hemorrhage that interrupted cerebral auditory pathways. The patient had a clear sensorium and showed no signs of epileptiform activity on EEG assessment. Lesion location was determined by magnetic resonance imaging (MRI), brainstem auditory evoked responses, and CT scanning.

By contrast, psychiatric symptomatology was reported by Trimble and Cummings (1981) in two cases of upper brainstem lesions. In one case, auditory hallucinations of a Schneiderian first-rank type were part of the psychiatric symptoms that followed a vascular abnormality in the region of the choroid plexus of the third ventricle. Neuropsychiatric symptoms, including auditory hallucinations, were present following idiopathic calcification of the basal ganglia in a case presented by Cummings, Gosenfeld, Houlihan, and McCaffery (1983). The patient had a 32 year psychiatric history with numerous hospitalizations. Despite this he had passed the bar exam, had superior level I.Q. scores and intact performance on Halstead-Reitan neuropsychological variables. Over time however, his psychometric performance declined so as to indicate intellectual deterioration and memory disturbance.

A more recent study by Lindstrom, Klockhoff, Svedberg, and Bergstrom (1987) showed 10 out of 20 inpatient schizophrenics to have abnormal auditory brainstem responses. Of the 11 patients admitting to auditory hallucinations 9 (82%) had abnormal brainstem responses. The authors conclude that auditory hallucinations may have an organic background.

It is evident from this review of the relationship of structural brain damage to auditory hallucinations that both cortical and subcortical brain systems may be implicated. At the level of the cortex, the temporal lobes appear to play a key role. They have been linked not only to auditory hallucinations, but also to olfactory and gustatory hallucinations (see Williams, 1968, 1969 for a review of temporal lobe syndromes). In epilepsy patients, auditory hallucinations are associated with stimulation of the first temporal convolution of either hemisphere (Penfield & Perot, 1963). Laterality effects are not clear in this population. Within psychiatric populations, where delineation of structural brain damage is still controversial, an association between left temporal lobe pathology and hallucinations is emerging (Takahashi et al., 1981).

Structural damage to subcortical brain systems can also result in auditory hallucinations, both in psychiatric populations (Lindstrom, Klockhoff, Svedberg, & Bergstrom, 1987) and normals (Cascino & Adams, 1986). Disruption of brainstem systems appears to be crucial in these groups, although the delineation of precise mechanisms is still unclear.

2.6 Visual Hallucinations

Complex visual hallucinations are usually associated with disturbances of posterior brain systems, specifically the temporal, temporo-occipital and parieto-occipital regions (Walsh, 1978). Simple visual hallucinations such as flashes of light, the appearance of colors or floating luminous points typically result from occipital lobe lesions (Walker & Cavenar, 1983). A wide array of both licit and illicit drugs have been causally linked to visual hallucinations (see Asaad & Shapiro, 1986; Cohen, 1984; Channer & Stanley, 1983; Siegel, 1977 for reviews). In addition, eye disease resulting in visuo-perceptual dysfunction has been linked to visual hallucinations (Berrios & Brook, 1984; Flach & Kaplan, 1983). Based on a series of 16 cases of tumors involving solely the optic nerves or chiasma, Weinberger and Grant (1940) concluded that a lesion at any level of the neuro-optic system could result in visual hallucinations.

Nevertheless, visual hallucinations are generally viewed as consequences of organic brain disease, rather than peripheral organ dysfunction (Roberts, 1984). Neurological events resulting in visual hallucinations may involve both cortical and subcortical brain regions, with or without psychiatric sequelae. Cerebral infarction, trauma, and tumor may all result in visual hallucinosis (Berrios, & Brook, 1984). Horrax (1923) reported on a series of 17 cases of complex visual hallucinations out of a pool of 873 verified cases of brain tumor. Postmortem data was available on six of these cases and all had circumscribed temporal lobe tumors that affected the optic fibres passing through the temporal lobes. None of the tumors encroached upon the occipital lobes. From the individual case reports cited, 9 involved the right temporal lobe, 3 the left temporal lobe, and on 5 no localizing data were available. A distinction was made between the simple, unformed visual hallucinations of occipital lobe tumors and the more complex, fully formed hallucinations associated with temporal lobe tumors.

Brust and Behrens (1977) described two cases of probable infarction of the posterior cerebral artery. The posterior cerebral artery is formed from the bifurcation of the basilar artery and it irrigates the medial and inferior surfaces of the temporal and occipital lobes (Carpenter, 1973). Both patients presented with scotomas and their complex visual hallucinations occurred in these blind spots. One patient experienced seizure activity, the other did not. No additional psychiatric symptoms were present in these cases and the authors concluded that occipital or occipito-temporal infarction was the most likely cause of the hallucinations.

Of a series of 120 cases of homonymous hemianopia (loss of sight in the same half of the visual field of each eye) reported by Kolmel (1985), 16 (13%) experienced complex visual hallucinations. Lesions were identified by CT scan, which showed the occipital lobe to be involved in all cases. In addition, lesions extended anteriorly to include portions of the ipsilateral parietal or temporal lobe (visual association cortex). In all cases subcortical involvement was present. Laterality of the lesion was not significant; 7 were left hemisphere, 8 were right hemisphere, and 1 was bilaterally distributed. Psychiatric or epileptic behaviors were absent in this group.

In contrast, of the 8 patients reported by Levine and Finklestein (1982), 7 experienced seizures and all 8 manifested signs of psychosis following right hemisphere stroke or trauma. All had sustained damage to a combination of the convex surfaces of the parietal or occipital lobes as assessed by clinical, EEG, and CT scan findings. Auditory and visual hallucinations were reported, as was delusional thinking in all cases. These findings are consistent with those of Price and Mesulam (1985) who described psychiatric symptoms emerging after unilateral right hemisphere infarctions.

Brief mention can be made of two subcortical syndromes that are characterized by visual hallucinations. These are peduncular hallucinosis and the Charles Bonnet syndrome. In peduncular hallucinosis, a lesion of the midbrain produces complex and vivid colored images of people or animals, accompanied by a state of confusion. The hallucinations are frequently "Lilliputian" in type and of many years duration. Most commonly they are found in the elderly and coexist with signs of visual impairment and vertigo (Berrios, 1985b; Walker & Cavenar, 1983). Hecaen and Albert (1978) note that they are most frequent late in the evening as the individual becomes drowsy and attention begins to wander. Dunn, Weisberg, and Nadell (1983) described a case of transient peduncular hallucinosis in a young child due to compression of the midbrain by a craniopharyngioma.

The Charles Bonnet syndrome is characterized by chronic visual hallucinations in the presence of severe visual impairment. Cognitive abilities remain intact and subjects are aware of the unreality of their visions. Confusion is absent, as are psychiatric symptoms (Hecaen & Albert, 1978; McNamara, Heros, & Boller, 1982; Rosenbaum, Harati, Rolak, & Freedman, 1987). The etiological mechanism for this disorder is unclear at this time.

As was discussed in the section on auditory hallucinations, direct electrical stimulation of the brain may induce hallucinations in epileptic patients. In the pioneering work of Penfield and Perot (1963), stimulation of the first temporal convolution of either hemisphere produced visual and auditory hallucinations. However, visual hallucinations were more strongly lateralized than auditory hallucinations and they showed a clear right hemisphere emphasis. They noted that the brain regions capable of eliciting visual hallucinations extended posteriorly, almost to the secondary visual association areas (Brodmann's area 19). They included the area that is devoted to speech in the left hemisphere. Findings were consistent both for patients who experienced visual hallucinations as part of their seizure activity and also for those patients in whom hallucinations were induced by direct electrical stimulation of the brain. While not discussed in the text it is evident from the authors' diagrams (see page 674, figure 13) that musical hallucinations, as well as visual hallucinations, were most frequently elicited from the right temporal lobe.

In a depth electrode study of 16 temporal lobe epileptics, Horowitz and Adams (1970) found no laterality effect for eliciting complex visual hallucinations. Ten of the 16 subjects reported visual hallucinations following stimulation. The principle areas linked to hallucinations were the posterior dorsal hippocampal region and the amygdala. The phenomenology of the hallucinations was similar to that reported by Penfield and Perot (1963).

In summary, lateralized damage to the cortex of the right hemisphere has been clearly linked to visual hallucinations. Thus while it is possible to hallucinate following left hemisphere damage, visual hallucinations are more likely to be found following right brain injury (Hecaen & Albert, 1978; Horrax, 1923; Lance, 1976; Penfield & Perot, 1963; Teuber, 1962). Laterality effects following subcortical brain stimulation or damage are less clear (Kolmel, 1985; Horowitz & Adams, 1970).

A wide array of different causes ranging from drugs to brain damage can induce visual hallucinations. In the case of visual hallucinations, care should be taken to rule-out eye-disease as a cause (Berrios & Brook, 1984). The hallucination itself may be as simple as unformed flashes of light, or as complex as a scene from nature, with people and animals moving within it. With increased elaboration of the visual hallucination, it is thought that temporal lobe systems become more involved (Williams, 1968, 1969). Psychiatric symptoms may or may not coexist with visual hallucinations. Similarly, epilepsy may or may not be present (Kolmel 1985; Levine & Finklestein, 1982). At the level of cortical brain systems, visual hallucinations following brain damage are likely to involve occipital, parietal, or temporal lobe systems (typically right greater than left).

2.7 Behavioral Treatment of Hallucinations

Examination of the literature on the behavioral treatment of hallucinations reveals that cases generally involve chronic psychiatric patients who are on medications, usually of an anti-psychotic type (Faloon & Talbot, 1981). Treatment strategies are frequently behavioral, although there are reports of the use of psychotherapy and hypnosis (Levick & Tepp, 1985; Ortega, 1984; Steingard & Frankel, 1985). A recent clinical approach showing great promise in the treatment of auditory hallucination is that of unilateral occlusion of the ear by means of a plug. This is based on the research work of Green and his associates, who are investigating the role of speech comprehension in psychiatric disorders

(Green, Hallett, & Hunter, 1983).

However, at this time, behavior modification strategies are the most common non-pharmacological intervention. Typically, several simultaneous strategies are applied to each patient. These involve exposure based techniques such as systematic desensitization, contingency management procedures, which are essentially operant conditioning approaches, cognitive behaviour therapy, after the style of such practitioners as Beck, Ellis, and Meichenbaum, assertive and social skills training and self control methods such as biofeedback (Johnston, 1984). Due to the overlapping use of behavioral technologies, studies have been categorized according to what is judged to be the prime behavioral intervention used to treat the patient's hallucinations.

Of four studies using exposure as a primary strategy only one used graded exposure alone (Slade, 1972). The others used systematic desensitization plus in vivo exposure to the hallucinatory provoking stimuli (Slade, 1973), satiation (Glaister, 1985), and graduated self-production and dismissal of the noxious stimuli (Fisher & Winkler, 1975). In all cases the symptom of hallucinations was alleviated and remained so upon follow-up, which ranged from 5 weeks to 5 years. Three of the four subjects were on medications during treatment and in all cases it was reduced substantially as therapy progressed. It was evident that in vivo exposure to stressful situations produced a more rapid decline in hallucinatory frequency than imaginal exposure (Slade, 1972, 1973).

A study by Nydegger (1972) utilized assertiveness training in combination with systematic desensitization and a verbal conditioning paradigm to extinguish auditory and visual hallucinations in a hospitalized paranoid schizophrenic patient. Following a two week baseline, treatment was instituted and all hallucinatory activity was eliminated within two months and remained so at follow-up two and a half years later.

The most commonly used strategies in the behavioral treatment of hallucinations are contingency management procedures. These involve the manipulation of the consequences of hallucinatory behavior and include a wide array of specific techniques. Examples are: timeout (Herron & DeArmound, 1978), aversion therapy (Bucher & Fabricatore, 1970), thought stopping (Samaan, 1975), tokens (Anderson & Alpert, 1974), stimulus interference (Alford & Turner, 1976), anger induction (Johnson, Shenoy, & Gilmore, 1983), self-monitoring and social reinforcement (Rutner & Bugle, 1969), contingency contracting (Alumbaugh, 1971), and covert assertion (Errickson, Darnell, & Labeck, 1978).

All the studies cited above describe single subjects, who were usually hospitalized, medicated and still hallucinated, despite a variety of pharmacological and psychotherapeutic interventions. Initially, baseline measurements of the frequency of hallucinations were taken and then a combination of behavior therapy techniques were implemented, sequentially and/or simultaneously. Several studies withdrew treatment and returned to baseline as part of their investigation. They showed an increased frequency of hallucinations during this period (Alford & Turner, 1976; Anderson & Alpert, 1974). Follow-up intervals ranged from 3 weeks to a year and in all but one case (Bucher & Fabricatore, 1970) subjects remained hallucination free.

While it is unclear which specific behavioral intervention was most effective in eliminating hallucinations, the research evidence to date does show that a wide variety of behavioral interventions can eliminate this distressing symptom. Studies used different subjects with different psychiatric diagnoses, both psychotic and non-psychotic, and a wide variety of interventions were implemented. Despite the heterogeneity of the subjects under study, certain common themes emerged. These involved reinforcing responses incompatible with hallucinatory behavior, improving the subjects' social skills, and promoting a sense of control over the symptom of hallucinations. It was noted by several authors that reduction of hallucinations was associated with improved social functioning (Alumbaugh, 1971; Burgio, Brown, & Tice, 1985; Rutner & Bugle, 1969). These ideas are supported by studies of self-control procedures spontaneously developed by hallucinated patients. They are reviewed in the following sections.

2.7.1 Self-Help Strategies

A comprehensive analysis of self-help strategies developed by hallucinators was undertaken by Falloon and Talbot (1981). Subjects were 40 chronic schizophrenic out-patients who experienced auditory hallucinations daily. They were interviewed over a six month period in order to determine what coping strategies they had developed to deal with their hallucinations. Thirty eight of the subjects were medicated (the other two failed to respond to medications) and on average, they had experienced their voices for 8.4 years (range 2 - 33 years). Coping strategies were classified by the authors into a tripartite system involving behavior change, alteration of physiological arousal, and cognitive methods.

Examples of behavior changes included: lying down, going for a walk, seeking out or withdrawing from interpersonal contact and taking drugs, both licit and/or illicit. Levels of physiological arousal were increased by exercise or loud music and decreased by deliberate relaxation. Additionally, sensory input was reduced by blocking the ears or closing the eyes. Cognitive changes were effected by such strategies as: actively listening to the voices and reflecting back the contents of the hallucinations as thoughts, ignoring the voices, trying to empty the mind of thoughts, arguing or debating with the voices and finally listening attentively to the voices and seriously considering their content.

The hallucinators who were least distressed by their voices were those who utilized a variety of different coping strategies, used them systematically and were cognizant of the environmental conditions that typically resulted in their hearing voices. Variables such as: sex, age of onset of hallucinations, subtype of schizophrenia, race, or type of medications were unrelated to ability to cope with voices. It was noted however, that less depressed subjects were more accepting of their voices. While no novel self-help strategies emerged from the Falloon and Talbot survey, it was evident that patients were applying the same kinds of behavioral treatments to themselves as were clinicians in hospital settings.

A second survey-study of self-control techniques in psychotic disorders by Breier and Strauss (1983) showed similar results to the Falloon and Talbot (1981) findings. While not specifically focused on hallucinators, 5 out of their 20 patients had hallucinations as a target variable. Subjects exerted control over their psychotic symptoms in three stages: first by becoming aware of their behaviors by self-monitoring, second by evaluating the consequences of their observations (i.e., the antecedents to their psychotic behaviors) and third by the use of self-control procedures. These included self-instruction, literally self-talk, reducing involvement with the surrounding environment and the converse, increasing involvement with the surrounding environment. Subjects who were unable to control their behaviors were those who failed to self-monitor and evaluate their behaviors. Four out of the five hallucinators were able to control their voices by modifying their activity levels.

It would seem that elements common to successful strategies for dealing with auditory hallucinations are: self-monitoring of behavior, evaluation of consequences, awareness of which situations are likely to induce hallucinations, and the systematic application of a variety of strategies to deal with the voices. The consequence of these behaviors is to increase the subjects' perceived control over their hallucinations, which in turn reduces their anxiety level. Reports from patients in the two studies cited indicate that strategies worked best when applied promptly after becoming aware of the voices or their precursors.

The idea of increased control over intrusive stimuli as a means of treating hallucinations can apply to visual as well as auditory stimuli. In two cases treated by the author, clients were taught how to deliberately induce and modify their visual hallucinations. As in the studies cited above, baseline frequency measures were taken and the antecedent and eliciting stimuli determined, as well as the social consequences. One client experienced visual hallucinations of rats, the other a visual image of the back of a man's head with a gun pointed at it. In both cases substantial anxiety was generated in the presence of the hallucinations, which persisted despite psychotropic medications and in one case a course of electro-convulsive therapy. The clients were taught to control discrete aspects of their visual hallucinations. This resulted in less anxiety in the presence of the hallucinated images, which rewarded further attempts at control of the images. Both clients completely eliminated their hallucinations. Follow-up, eighteen months later showed that both subjects to be hallucination free.

2.7.2 Auditory Occlusion Strategies

An alternate method of treating auditory hallucinations that has been employed spontaneously by schizophrenic patients and developed into a more specific technique by clinicians is that of using earplugs. A case report by McGuffin (1979) described a hallucinated schizophrenic who achieved temporary relief by stuffing his ears with cotton wool. Following this Green (1979) and Green and Kotenko (1980) suggested that occlusion of the ear with poorest speech comprehension might prove beneficial in the treatment of auditory hallucinations.

A series of four case studies (Done, Frith, and Owens, 1986; James, 1983; Birchwood, 1986; Levick & Peselow, 1986) have since demonstrated the value of a unilateral earplug in reducing the frequency and intensity of auditory hallucinations. In all cases subjects were heavily medicated schizophrenics (3 chronic schizophrenics, 2 acute schizophrenics; 4 males, 1 female) whose voices proved refractory to drug interventions. Unilateral auditory occlusion dramatically reduced auditory hallucinations in all cases. However, the choice of which ear to occlude appears to be problematic.

Green and Kotenko (1980) recommend plugging the ear with inferior comprehension as assessed by the Auditory Comprehension Test (ACT), a measure of short story recall that is administered under both monaural and binaural conditions. Of the four studies cited, two (Done, Frith, & Owens, 1986; Levick & Peselow, 1986) found better results by plugging the ear showing superior speech comprehension. One (Birchwood, 1986) plugged only the poorest ear, with excellent results and in the study by James (1983) mixed results were found. For the acute female schizophrenic in his study, best results were obtained by plugging the left ear, while in the case of the male chronic schizophrenic, best results were obtained after plugging the right ear. It is unclear if an ACT was given to this subject and it was noted that in both cases switching the plug to the other ear resulted in a continued but slowed rate of improvement. Follow-up periods in the James (1983) and Birchwood (1986) studies ranged from six months to three years, with positive results. The patient in the Levick &

Peselow (1986) study relapsed after discharge and follow-up is unclear in the paper by Done, Frith, & Owens (1986).

Further studies are needed to determine which groups of hallucinated patients are best suited to this treatment modality. At present, the technique appears promising, is non-invasive, works promptly when appropriate, and does not produce side-effects. Why or how a unilateral earplug is effective remains controversial and it is noted that requiring the patient to verbalize aloud during their hallucinations helps potentate the benefits of the earplug treatment (Birchwood, 1986).

More conventional psychotherapeutic techniques have also been used in the treatment of refractory hallucinations. Steingard and Frankel (1985) report the successful treatment of auditory and visual hallucinations in a 17 year-old girl by means of hypnosis. The patient was taught to use self-hypnosis as a method of controlling her hallucinations. Ortega (1984), utilized hypnotic trance to allow a 21 year-old college student to safely reexperience frightening hypnopompic hallucinations. Hypnosis was used to facilitate relaxation, so that the fear inducing images could be reciprocally inhibited. In both of these studies subjects were free of chronic mental illness and not taking therapeutic levels of psychotropic medication.

To summarize this section on the non-pharmacological treatment of hallucinations, it can be seen that the bulk of literature reviewed involves the application of various behavioral technologies in each study. Behavior therapies can be categorized according to their principal mode of action: exposure based, contingency management procedures, cognitive approaches, assertive and social skills training and the catchall category of self-control procedures. Of the behavioral studies cited, all resulted in improvement, that is a decrease in the frequency and intensity of the patient's hallucinations. No specific behavioral intervention was shown to be superior; in fact, best results were achieved when several different techniques were used with the same client. In addition, prompt and systematic application of behavioral interventions in subjects who recognized the situations that were most likely to elicit hallucinations promoted good results.

Comparison of hospital based behavior therapy and techniques evolved by patients living in the community showed a high degree of overlap. Similar strategies worked with other psychotic behaviors as well as hallucinations (Breier & Strauss, 1983) and while these are most commonly used with auditory hallucinations, the author has experience using behavioral interventions with visual hallucinations. It was noted that improving social skills was associated with a reduced frequency of hallucinations (Nydegger, 1972) and the corollary was also true, reduced frequency of hallucinations was linked to improved social skills (Alumbaugh, 1971; Burgio, Brown & Tice, 1985; Rutner & Bugle, 1969). At a cognitive level, the perception of control of the voices was important to patients and this led to a reduction in the amount of distress generated by the hallucinations.

An alternate method of treating hallucinations involved unilateral auditory occlusion, that is plugging one ear. Of the published studies, all found the procedure effective, although which is the best ear to plug remains unclear. Hypnosis has also been used successfully in the treatment of hallucinations, but the cases cited dealt with less severely disturbed subjects than the behavioral and earplug studies.

2.8 Pharmacological Treatment of Hallucinations

Hallucinations do not typically occur in isolation, they are usually found embedded in a complex of symptoms that, together, constitute the basis for a diagnosis of a mental illness. Because of this, little has been written specifically on the topic of psychopharmacological interventions for hallucinosis. Individuals thought to be acutely psychotic are typically treated with neuroleptic drugs that relieve not only hallucinations, but also additional symptoms such as: agitation, thought disorder, hostility, delusions, and incoherence (Ellison, 1985).

Should hallucinations persist in spite of anti-psychotic drug therapy, there is evidence that the addition of benzodiazepines may prove beneficial. Two drugs reportedly useful in the treatment of hallucinations, as found in mental illness, are diazepam and estazolam. The former, more commonly known as Valium, was reported by Kramer (1967) to relieve chronic auditory hallucinosis in four out of five patients being followed-up in a state hospital aftercare clinic. These medicated patients had experienced hallucinations from 3 to 18 years. The author reports that while phenothiazines had helped the patients in other spheres, the addition of Valium specifically reduced the presence of auditory hallucinations. A more recent note by Jos, Schneider, and Gannon (1985) reports two cases of improvement of auditory hallucinations with the addition of diazepam to the existing drug regimen. In one case, visual hallucinations were also eliminated and the authors indicate that significant anxiety component was present in both cases.

A second drug reported to have specific antihallucinatory properties is the triazolobenzodiazepine, Estazolam, an anti-anxiety agent that is currently not available in North America. In a multi-centre, double-blind, cross-over study by Lingjaerde (1982), Estazolam, in addition to neuroleptic medication, was found to be superior to placebo in improving hallucinations (auditory and visual) and patients' overall mental state. The patient group (N = 51) was composed primarily of medicated schizophrenics all of whom were experiencing auditory hallucinations. Most had been ill for over three years and were hallucinated throughout their illness. The diagnostic criteria were those of Leonhard (1979) which are used in Europe, but rarely in North America.

This study was followed-up by the work of Astrup and Vatten (1984) who also used a combination of Estazolam and neuroleptics with hallucinated schizophrenics (N = 53). As above, the diagnostic classification was that of Leonhard and 25 of the 53 patients (47%) were judged to have benefited from the drug combination. Specific criteria for improvement are not listed.

Taken together, these four studies suggest a role for the benzodiazepine drugs in the treatment of chronic hallucinations, as found in medicated schizophrenics. Both classes of drugs are used with caution, due to serious potential side-effects, these being tardive dyskinesia with the phenothiazenes (Schatzberg & Cole, 1986) and dependence with the benzodiazepines (Lader, 1987). It should also be noted that psychoactive drugs used to treat mental illness may themselves induce hallucinations. For example, the tricyclic antidepressant imipramine has been shown to induce visual hallucinations (Klein, 1965; Schlauch, 1979) as has amitriptyline (Hemmingsen & Rafaelson, 1980) and the unicyclic antidepressant, bupropion (Johnston, Lineberry, & Frieden, 1986). An extensive list of prescribed drugs that may induce hallucinations is provided by Estroff and Gold (1986).

Chapter III

Review of the Literature: Part II, Theories of Hallucinations

3.1 Introduction

Theoretical explanations of hallucinations are divided into two basic categories, neuropsychological models and cognitive models. The neuropsychological models tend to be more general in scope, offering a comprehensive overview of an array of hallucinatory experiences. Cognitive models are more focused in nature, tending to examine specific types of hallucinations or hallucinators (Hoffman, 1986). The purpose of this section is to examine the more explicitly stated theoretical conceptions of hallucinations. The neuropsychological models are addressed first, then the cognitive models. The literature on speech dysregulation and hallucinations is reviewed separately.

3.2 Neuropsychological Models

West (1962, 1968, 1975) has proposed a comprehensive model of the hallucinatory experience. This is based on two basic assumptions. First, that the personal experience of the individual affects the brain in such a way as to leave a permanent neural record. Ideas and images are derived from these traces which provide a neurophysiological substrate for memory, thought, imagination, and fantasy. The role of the limbic system in the affective coloring of intellectual and perceptual functions is stressed (MacLean, 1955). This allows for an interaction of perception and emotion that occurs primarily at an unconscious level. Conscious awareness is mediated neuropsychologically by a general arousal system involving the ascending midbrain reticular activating system. This is not meant to imply that other brain systems are functionally irrelevant, see for example Williams' discussion of the temporal lobes (Williams, 1968).

West's second basic assumption is that the construct personality, may be viewed as the dynamic interaction of psychobiological forces of internal and external origin to the individual. These psychobiological forces may usefully be conceptualized as transactions between systems. A balance exists between innate, internal forces and external, environmental forces. The effect of this interaction is to have an organizing effect on the constant flow of information impinging on the individual. This in turn modifies memories, dreams, ideas, fantasies or hallucinations and the affects associated with them. The result is that cultural and psychodynamic factors play a significant role in determining the form and meaning of hallucinations (Bourguignon, 1970; Modell, 1960; Weinstein, 1970).

Typically, the individual receives vastly more sensory input than he or she could possibly be aware of, let alone consciously process. Information is actively excluded from awareness by the brain and only limited fields of awareness are allowed. This scanning and screening process (concentration) keeps unwanted and irrelevant information out of consciousness. Not only external sensory inputs are screened, so too are internal sensations, previously stored memories and their elaborations. The normal flow of information experienced while awake serves to maintain scanning and screening functions.

Should sensory input be reduced or impaired, its organizing effect on the scanning and screening mechanisms is decreased. Such events may be accompanied by a parallel decrease in the stimulating effects of the ascending reticular activating system. This leads to diminished arousal and awareness. However, should sensory input be reduced or distorted and cortical arousal maintained (i.e., awareness) the normal flow of information no longer serves its inhibitory function. Consequently perceptual traces may be "released" and reach conscious awareness as new, familiar or unusual mental experiences. In order to reach consciousness with hallucinatory vividness two conditions need to be fulfilled.

Firstly, there must be a sufficient general level of arousal for awareness to occur. Secondly, the neural substrate of the emerging percept must "fire" and "reverberate" sufficiently intensely to command awareness. This can be accomplished by either a reduction of normal inhibitory mechanisms or by direct stimulation of the brain, as in cases of temporal lobe epilepsy (Penfield & Perot, 1963). Part of the normal pattern of inhibition is a steady flow of information to the brain from the internal and external environment. Distortion of this information flow, in a context of cortical arousal sufficient to provide awareness, will result in this material occupying the field of awareness. West's theory states that both a sustained level and variety of sensory input is necessary to inhibit percepts or memory traces from reaching awareness. Should attention commanding sensory input fall below a certain level, previously recorded perceptions may emerge into consciousness due to disinhibition of the cerebral systems that represent them. Sufficient cortical arousal is necessary for this to occur and the greater the level of arousal the more vivid the perception. This process can result in fantasies, illusions, visions, dreams or hallucinations. As an analogy to help visualize these processes West (1975) provides the following:

> A man is standing at a closed, clear glass window opposite a fireplace, looking out at his garden in the sunset. He sees only the outside world. As it becomes darker outside, however, images of the objects in the room behind him can be seen reflected dimly in the window glass. For a time he may see either the garden (if he gazes into the distance) or

> the reflection of the room's interior (if he focuses on the glass a few inches from his face). Night falls, but the fire still burns brightly in the fireplace and illuminates the room. The watcher now sees in the glass a reflection of the interior of the room behind him, which appears to be outside the window. This vision becomes dimmer as the fire dies down, and, finally when it is dark both outside and within, nothing more is seen. However, if the fire flares up from time to time, the visions in the glass reappear.
>
> In perceptual release, the daylight (sensory input) is reduced while the interior illumination (general level of arousal) remains bright, and images originating within the rooms of our brains may be perceived as though they came from outside the windows of our senses (pp. 301-302).

In summary, West's (1962, 1968, 1975) theory of perceptual release proposes that effective sensory input functions to inhibit previously encoded percepts from reaching consciousness. In the context of a sufficient level of arousal to permit awareness, impairment of adequate sensory input results in previously encoded percepts being released into awareness. These may be experienced as hallucinations. Ways in which adequate sensory input can be impaired include: reduction in the absolute level, as for example, during sleep, by means of input overload as in delirium, and by reduced psychological contact with the environment, in for example, the dissociative states.

To illustrate his theory, West describes the process of falling asleep. During this process an individual loses awareness of the environment. Simultaneously, cortical arousal may decrease less rapidly and still allow some appreciation of reality. Hypnagogic phenomena can occur at this time. As further contact with the environment is lost sleep begins. External or internal stimuli interact with the basic ninety minute sleep-state cycle to bring the individual into a zone of spontaneous perceptual release, where dreaming occurs. During the process of awakening, the sleeper again passes through a zone of perceptual release, with increased dream intensity and possible hypnopompic hallucinations upon awakening.

For West, the relationship between arousal and the ability of the brain to process information is essentially an inverted U shaped function. There is an optimal balance between level of arousal, as mediated neurophysiologically and a state of maximal concentration. As an individual moves from low to high arousal he or she passes from deep sleep to dreaming to waking, where hypnagogic phenomena may be experienced. Then, once awake in a state of relaxation, increased arousal can lead to an alert but calm state and then on to one of maximum concentration. With further increase of arousal the individual becomes alert but tense, then distractible and restless. With even more arousal dissociative phenomena may occur, followed by psychotic excitement, delirium, convulsions and coma (West, 1962).

A variety of situations conducive to hallucinations are described by West within the context of perceptual release theory. These include: sensory isolation experiments, periods of prolonged wakefulness, mystical experiences, hypnosis, sensory monotony, impairment of sensory input (e.g., cataracts or phantom limb phenomena) and, drug induced hallucinations. The hallucinations of the mentally ill are conceptualized within the framework of the mechanism of dissociation, as well as perceptual release theory. Dissociation is a psychodynamic defence mechanism whereby conflicting impulses are prevented from reaching consciousness. Disorders typically associated with this process are psychogenic amnesia, fugue states, somnambulism, and multiple personality (Goldenson, 1984; West, 1975).

Perceptual release theory has its roots in the work of the English neurologist Hughlings Jackson, who considered that hallucinatory phenomena resulted from the failure of cerebrally mediated inhibitory mechanisms (see Taylor, Holmes, & Walshe, 1958). The role of disrupted sensory input in the occurrence of disinhibition phenomena is attributed to Evarts (1957) whose work involved the effects of LSD on animals. West (1962) further elaborated these ideas into a general theory of hallucinatory phenomena. In essence, his theory postulates that a sufficient variety and intensity of sensory input is necessary to prevent previously encoded percepts or memories from reaching conscious awareness. A failure of sensory input sufficient to hold attention may lead to a release of these memory traces due to disinhibition of the cerebral systems that represent them. If this occurs in the presence of cortical arousal, the released perceptions may reach conscious awareness to be reexperienced as fantasies, illusions, visions, dreams or hallucinations. The model is based upon the assumptions that the brain constantly records sensations and experiences and that this processed sensory data is available as a source of ideas and images. In addition, a constant interplay is present in the brain between previously encoded information and fresh incoming sensory data. The input of new information serves to inhibit the release of previously recorded material to conscious awareness. Thus a balance is maintained between internal and external levels of sensory input. Levels of arousal are mediated primarily by subcortical brain systems (ascending midbrain reticular activating system) while the cognitive elaboration of the hallucinatory experiences reflects the input of cortical association areas (Marrazzi, Woodruff, & Kennedy, 1972).

3.3 Neurophysiological Models

West's model of perceptual release may be viewed as a psychological description of neurophysiological processes. Other more specifically neurophysiological theories have been developed by Evarts (1962) and Scheibel and Scheibel (1962, 1970). In the tradition of Hughlings Jackson, Evarts (1962) proposed that, "Some hallucinations are correlative with a neurophysiologic process similar to one which occurs during sleep, and which consists of abnormally brisk discharge of centres involved in waking perception" (p. 9). He then cites evidence relating to increased cortical excitability during sleep and its association with dream states. An optimal level of disinhibition is deemed necessary for hallucinations or dreams to occur. This usually occurs during light sleep. With deeper sleep, the random activities of the cortex are too depatterned to produce meaningful thoughts or imagery. With increased levels of inhibition the waking state is maintained.

Evarts based his hypothesis about the nature of hallucinations on three main ideas. First, that dreams and hallucinations are similar in nature. Thus there should be an association between the neurophysiology of dreaming and hallucinations while awake. Second, that both dreams and hallucinations are linked to the cerebral systems involved with waking perception. This would imply that the study of normal perceptual mechanisms (e.g., vision) would be relevant to the underlying mechanisms of visual hallucinations. Third, hallucinations, dreams and normal perceptual processes reflect a "strong discharge" of the same cerebral mechanisms in whom a "weak discharge" would be equivalent to thoughts, ideas, or memories. The idea of strength of neuronal discharge as the source of hallucinatory activity is attributed to Hughlings Jackson, who believed that it resulted from a loss of inhibition. This would occur normally in the process of falling asleep or pathologically in the case of cerebral disease (Taylor, Holmes, & Walshe, 1958). Evarts believed that the crucial neural systems for modulating levels of disinhibition resided in the brainstem. The work of Scheibel and Scheibel (1962, 1970) has continued investigation into the role of the brainstem reticular formation in cerebral processing.

Citing evidence that hallucinations can result from a variety of pathologies at many levels of the nervous system, the Scheibel's (1962) emphasize the role of brainstem mechanisms in monitoring data from divergent parts of the brain. The reticular formation is a ubiquitous system that receives a constant flow of afferent and efferent sensory-motor information that is reflective of the organism's internal and external environment. In addition, it projects to many regions of the cortex (via the ascending reticular activating system) and so is in a position to influence information analyzed by higher association areas. Because of its biasing influence on sensory data at so many levels of analysis, Scheibel and Scheibel (1970) propose that the reticular formation plays a crucial role in the organism's experience and interpretation of reality. They suggest that pathological functioning of the reticular formation could result in decreased weighting of valid external sensory information and overelaboration of internal sensory data, resulting in delusions and hallucinatory misperceptions (Scheibel & Scheibel, 1962).

3.4 Summary

The most completely developed model of hallucinations is the perceptual release theory of West (1962, 1968, 1975). He proposes that hallucinations result from an imbalance between effective sensory input and disinhibition of previously encoded perceptual material. The normal flow of sensory input in the context of arousal mediated by the ascending reticular activating system is sufficient to inhibit perceptual release. However, should sensory input decrease while cortical arousal remains high, sensory information that is normally inhibited may reach consciousness. Optimal arousal is conceived of as an inverted U function, with a continuum of low to high arousal states on either side.

The model of Evarts (1962) is based on ideas of the English neurologist Hughlings Jackson who suggested that hallucinations result from strong neuronal discharges due to a failure of inhibitory mechanisms. That is, hallucinations are a release phenomenon. Evarts proposed that dreams and hallucinations are similar in nature and that they are linked to normal perceptual mechanisms. Also that thoughts, ideas, and memories, as well as hallucinations, dreams and waking perceptions all reflect weak and strong neuronal discharges of the same cerebral systems. The role of brainstem nuclei in inhibiting peripheral receptor and cortical receiving areas is stressed.

The work of Scheibel and Scheibel (1962, 1970) emphasizes the role of the brainstem reticular formation as a nonspecific system for the biasing of attention and arousal. This system receives a constant flow of incoming and outgoing sensory-motor data which it samples, modulates and transmits to the cortex via thalamic nuclei, or sends to the periphery by means of the reticulospinal tracts. As a monitor of all information passing up and down the neuraxis, Scheibel and Scheibel suggest the brainstem plays an important role in the organism's interpretation of reality. Abnormalities of reticular formation function could alter the balance between internal and externally generated sensory information, resulting in perceptually anomalous experiences.

3.5 Cognitive Models 1: Horowitz

An information processing model of hallucinations has been proposed by Horowitz (1975a, 1975b). This is a general theory of hallucinations, with an emphasis on the visual system, whereas the models of Slade (1976a) and Hoffman (1986) refer specifically to auditory hallucinations. For Horowitz, hallucinations are images based on internal sources of information that are perceived "as if" the information came from sources external to the organism. There is a discrepancy between the individual's experience and reality, such that the hallucinated image appears to have entered the mind without conscious intent. The term image is used to mean a mental representation of some previous sensory experience that is recalled without external stimulation (Goldenson, 1984).

Images may be categorized according to intensity or vividness, by the context in which they are experienced, by their content and by their independence from or degree of interaction with the external environment. Traditionally, hallucinations are defined as occurring in the absence of external environmental stimuli. However experiences such as illusions, synesthesia, perceptual distortion, deja vu, negative hallucinations and afterimages all involve incorporation of varying degrees of environmental information. To summarize, for Horowitz, hallucinations are defined as images that are based on internally derived information which is erroneously attributed to external sources and which intrudes, unbidden, upon awareness.

The model postulates four basic dimensions of the hallucinatory experience. These are: an image system of representation, an intensification of input into the image system, impaired information processing of images, and alterations in the cognitive control over image formation (Horowitz, 1975b). Hallucinations are conscious images that are derived from internal sources of information and are incorrectly judged to be of external origin. They typically occur without conscious intent or the realization that one is thinking rather than perceiving. Each dimension will briefly be discussed in terms of its contribution to the hallucinatory experience.

Horowitz suggests that hallucinations are a form of thinking and that thought can be separated according to its mode of representation into enactive or motor thought, lexical thought, and images. Images may occur in different sensory modalities. Normal thinking incorporates a blend of all three types of thought. Activation of the image system relative to normal levels of lexical or enactive thought may be one way to predispose an individual towards hallucinations. For example, electrical stimulation of the visual system results in the perceptual experience of flashes of light, geometric shapes, or even faces (Horowitz & Adams, 1970).

Should one system of image representation be heightened relative to others, the resulting construct could take on a quasi-perceptual quality due to its intensity. If this process of intensification were to interfere with the normal translation of images into lexical thought, the unusual nature or "strangeness" of these thoughts would be increased. Similarly, if an image of unusual vividness is experienced in an infrequently used modality, this would also accentuate its unfamiliarity. For example, it is likely that an individual who infrequently utilized visual imagery and suddenly experienced powerful visual images would react strongly to the strangeness of this mental experience. Horowitz's model proposes that accentuated use of an image system of representation is one determinant of the hallucinatory experience. Individuals unaccustomed to this process would be more likely to react to the experience as if it were perceptual in nature.

A second dimension of the hallucinatory experience involves relative intensification of internal input into the image system of representation. Normal cerebration is a blending of internal and external sources of information to form images. This necessitates dual input models of image formation (West, 1962). These have been shown to have a degree of reciprocal inhibition within modalities, such that as awareness of internal sources of information increases, awareness of external sources of information declines. Dual input models postulate that a sustained level and variety of stimulation is necessary to inhibit the emergence of internally generated information into consciousness (see previous discussion of perceptual release theory by West).

Horowitz describes five conditions under which relative over activation of internal sources of information could occur. These include reduction of external input, with no relative lowering of the activity of the representational system. An example of this would be sensory deprivation hallucinations. Increasing activation of the representational system, without an increase in the flow of externally sourced information, as in direct brain stimulation hallucinations. Augmentation of internal input due to the arousal of ideas and feelings derived from intense need states (e.g., bereavement hallucinations). A reduction in normal levels of inhibition of internal input as occurs in dreams and hypnagogic imagery. Lastly, a condition in which internal sources of information occupy systems designed to analyze perceptual (i.e., external) material. This can occur when an unclear perceptual source is elaborated into an image congruent with the organisms internal need state. A frightened sentry on night duty may "see" a distorted tree or shadow as the enemy approaching.

The third dimension in Horowitz's model involves faulty information processing of formed images. This is an incorrect elaboration of what Horowitz calls the baseline image. In order for images to be fully processed, comparisons with internal and external sources of information are necessary as well as transformation of the image by the lexical system. Images are evaluated in terms of past experiences, familiarity and reality. Disruption of this information processing cycle would lead to images being conceptually isolated and acquiring perceptual rather than thought like characteristics. Stimuli that are brief, novel, and ambiguous would give rise to images that are hard to process and would therefore tend to be misinterpreted.

Impairment of information processing could result from cognitive impairment, psychological conflict or states of high arousal. Disruption of the transformation of an image into word representation due to cognitive impairment would likely lead to an unusual subjective experience of the image. Similarly, psychological defence mechanisms may act by interfering with information transformation. For example, repression and denial can be conceptualized as the inhibition of entry into a system of image representation. In intense need states, reality may be selectively ignored and an image accepted as if it were real, in order to sustain psychological hope.

The fourth dimension of Horowitz's model has to do with the intrusion of internally generated images into conscious awareness. This involves a loss of control and the unplanned nature of the intrusion serves to distance it from normal thought processes. Circumstances leading to the emergence of such images would include stressful perceptions resulting in flashbacks, combat nightmares, or bereavement hallucinations. Normally warded-off ideas or feelings that are threatening to the individual and reach conscious awareness are also likely to be perceived as foreign to the individual. Threatening ideas or feelings are routinely inhibited, but in the event of failure of inhibition, these partially processed internal events can intrude upon awareness unbidden. A third circumstance leading to intrusive images involves the transformation of affective states. Hallucinations, as a form of thought, may represent emotional states. A guilty person may hear accusatory voices, while a depressed individual may hear derogatory voices. However, in addition to mirroring an affective state, Horowitz suggests that hallucinations may occur in order to alter unpleasant emotions. Thus a person in despair may hallucinate an image of a lost object. The above are examples of undercontrol of internal input into image formation systems resulting in intrusive experiences. They may result from reliving incompletely processed stressful perceptions, the reemergence into consciousness of threatening ideas or feelings, and from the need to transform excessively distressing emotional states.

In summary, Horowitz (1975a, 1975b) proposes four principle dimensions to account for the hallucinatory experience. Firstly, an image system of representation for sensory experiences. Secondly, internal input into an image system of representation. Thirdly, impaired information processing of incompletely formed images, and fourthly, loss of control of internal input into the image system of representation. Hallucinations are intense images of internal origin that are erroneously attributed to external sources and intrude, unbidden, into awareness. Relative over activation of an image system, relative to other modes of information processing (e.g., lexical or enactive) can lead to a quasi-perceptual rather than thought-like quality to this mental content. Thinking is typically a blend of differing styles of information processing and vivid experience of one mode relative to another would be experienced as strange or unusual by the individual.

Normal cerebration also involves blending information of internal and external origin in image form. Intensification of internally derived information disturbs this balance and may result in internally generated material becoming conscious, but being reacted to, "as if" it originated externally. Ways in which this could happen include: reduced external input with no change in the level of internal input, increased activity of the representational system with no change in external levels of input, increased internal input as a result of emotional arousal, reduced inhibition of normal levels of internal input and finally conditions in which internal inputs occupy information processing channels normally utilized by material of external origin.

Impaired processing of formed images involving comparisons between internally and externally generated information can lead to the conceptual isolation of an image. During the elaboration of an image it is compared with past experience, familiarity and reality. Novel, brief and ambiguous stimuli would all be hard to process and are therefore likely to be misinterpreted. The last dimension of Horowitz's model involves loss of control over internal inputs into the image system of representation. This involves reliving stressful events (flashbacks) and normally suppressed painful emotions. The intrusive nature of these experiences makes them seem foreign and strange to the individual and unrelated to ongoing thought processes. Hallucinations are regarded as a final common pathway expressing combinations of Horowitz's four dimensions.

3.6 Cognitive Models 2: Slade

A speculative model of auditory hallucinations has been proposed by Slade (1976a). It consists of four factors: stress events, a hallucinatory predisposition, levels of external stimulation, and mood state improvement. Slade hypothesizes that auditory hallucinations result from an interaction of psychological stress with a genetically determined predisposition to hallucinate. External stimulation is conceived of as a variable that serves to inhibit auditory hallucinations, while mood state improvement following a hallucinatory experience may act to positively reinforce the preceding hallucinatory event. Thus, the model involves constitutional variables (predisposition), environmental variables (stress, levels of stimulation), and learning variables (positive reinforcement). Slade (1976a) emphasizes the speculative nature of his model and offers it as a means of conceptualizing and expanding current findings in the area of hallucinations. The model is based on experimental and clinical work (Slade, 1972, 1973, 1974, 1976b) that revealed a relationship between the occurrence of auditory hallucinations and a self-reported, generalized disturbance of mood.

The first factor in Slade's model is that of psychological stress. Stress is defined as, "any event, of either external (situational) or internal (cognitive) origin, which gives rise to a relative elevation and disturbance of mood state, specifically of tension and anxiety" (Slade, 1976a, p. 417). This is a response-based definition of stress, that allows for individual differences in reacting to stressful events. It is not meant to imply cortical or autonomic arousal, rather it reflects the individual's self-report. Stressors may be discrete or chronic and may originate internally or externally. Slade argues that increased stress levels raise the probability of a hallucinatory experience.

A second factor relating directly to the probability of experiencing hallucinations is the predisposition to hallucinate. This involves a combination of vivid mental imagery and poor reality-testing in the auditory modality. In the context of psychologically stressful events (factor I) vivid mental imagery is triggered. This is experienced as a hallucination because of poor reality testing. Reality testing involves the objective evaluation of sensory impressions which allow the individual to distinguish between the internal and external world. Additionally, it is predicted that the content of the hallucination should be related to the precipitating stress events. What is crucial is the interaction of the stressful events with the predisposition to hallucinate. Thus the prognosis of an individual who hallucinates in the presence of minor stressors is less favorable than that of an individual whose hallucinations are only triggered by severe stressors.

The third factor in Slade's model is that of external stimulation. Certain forms of external stimulation are proposed to have an inhibitory effect on auditory hallucinations. In a previous study Slade (1974) showed that the frequency of auditory hallucinations decreased as information processing demands increased. Using a technique suggested by the work of Antrobus (1968), on the relationship between rate of information processing and stimulus-independent thought, Slade (1974) tested two hallucinated schizophrenic patients under a variety of information processing conditions. He concluded that external stimulation that interfered with symptoms (i.e., stimuli which actively engaged the patient's attention) but did not interfere with task performance were effective in reducing hallucinatory frequency. A "limited channel capacity" model was suggested such that as patient's paid more attention to external sources of information and hence less to internal sources, the frequency of their auditory hallucinations declined. This process is similar to the dual-input information processing models proposed by West (1975) and Horowitz (1975a, 1975b).

The fourth factor in Slade's model is that of mood state improvement. This refers to self-rated mood state by the hallucinated individual. Slade (1972, 1973) noted that following hallucinatory experiences, patients' reported feeling better. He therefore suggested the possibility of an anxiety reducing effect for hallucinations that would serve to positively reinforce their further occurrence. This in turn results in a positive feedback loop that lowers the threshold for future hallucinatory occurrences. A response, ordinarily aversive in its own right (the hallucination) appears to be positively reinforced by its consequences (improved mood state). Slade suggests that in chronic schizophrenic patients who regularly hallucinate, the hallucinatory threshold has been so lowered that minimally stressful events can precipitate hallucinations. These hallucinations are seen as a learned response with factor I (stressful events) playing a very minor role.

Based on this model Slade suggests several different approaches to treatment. They include, attempting to control patients' responses to stress events (factor I) by means of pharmacological or psychotherapeutic interventions. Trying to control the occurrence of hallucinations by changing external levels of stimulation (e.g., diversional activities) or reinforcing behaviours incompatible with hallucinating (e.g., engaging in conversation). And thirdly by attempting to raise the patients' hallucinatory threshold (factor II) by replacing the positively reinforcing aspects of the hallucinatory act (improved mood) with negative reinforcement. This has been tried experimentally by means of aversion therapy (Alford & Turner, 1976; Anderson & Alpert, 1974; Bucher & Fabricatore, 1970). This latter approach would be most useful with chronically hallucinated patients.

To summarize Slade's (1976a) model, four factors are proposed to account for auditory hallucinations. Factor I is stress events, that is situational or cognitive occurrences that produce increased levels of self-reported tension and anxiety. Factor II is the individuals hallucinatory predisposition level, that is the threshold for hallucinations. This is genetically determined and involves vivid mental imagery and poor reality-testing in the auditory modality. Factor III is the level of external stimulation to which the individual is responding. Conscious awareness functions as a "limited channel capacity" (Antrobus, 1968) such that external stimuli are apprehended at the expense of internal events. Factor IV is a positive reinforcement effect. This involves the mood state improvement reported by patients following hallucinations and a possible reduction in the critical threshold to experience hallucinations.

The factors interact such that stressful events combine with a hallucinatory predisposition that is genetically determined. The role of external stimulation is to inhibit the conscious experience of hallucinations, while the improved mood state following hallucinations can be viewed as a positive reinforcer that acts to lower the threshold for future hallucinations. Slade stresses the speculative nature of his model, but argues that it leads to specific predictions and testable hypotheses and therefore has heuristic value. In fact, Slade and his collaborators have gone on to investigate specific aspects of his model in a series of studies (Bentall & Slade, 1985; Launay & Slade, 1981; Margo, Hemsley & Slade, 1981; Slade, 1974, 1976b; Young, Bentall, Slade & Dewey, 1986 & 1987).

3.7 Speech Dysregulation and Auditory Hallucinations

Hallucinated schizophrenics frequently hear voices that sound as if their thoughts were being spoken out loud (Fish, 1976). This observation raises the question of interaction between thought and language in schizophrenia and whether there is a mechanism whereby thoughts can be heard "audibly," but not spoken aloud. An approach to this problem has been through investigation of the phenomenon of inner speech, also known as endophasy, subvocal speech, silent speech, covert speech, or private speech (Garrity, 1977; Vygotsky, 1934/1962). It may be defined as a truncated version of social speech, involving peripheral speech-motor activity and characterized by attenuated movements of the speech muscles (Garrity, 1977). Inner speech functions to utilize language in the regulation of behavior (Meichenbaum, 1975).

Within the field of mental illness, initial speculation concerning the relationship between inner speech and auditory hallucinations is attributed to Kandinsky (1890) as cited by the Russian authors Bazhin, Wasserman and Tonokonogh (1975). In the English scientific literature, a series of investigations by Gould (1948, 1949, 1950) independently studied this problem.

In order to determine the possibility of a relationship between auditory hallucinations and covert speech mechanisms Gould (1948) compared electromyographic (EMG) data on 100 psychiatric patients and 100 controls. Recordings were taken from the lower lip and chin. Subjects did not know the purpose of the investigation, being told it was "something like a brainwave test" (Gould, 1948, p. 368). Patients were a heterogeneous group containing hallucinators, non-hallucinators and those unknown with respect to hallucinations. Diagnostically the patient group was also heterogeneous, with an emphasis on schizophrenics (72/100 cases) but also including alcoholics and organic brain syndromes. Control subjects were recruited from hospital staff and students. Results showed that 28% of the normals and 58% of the patients had an increase in muscle tension in the vocal musculature as defined by EMG recordings. A comparison of hallucinating versus non-hallucinating patients revealed that 83% of the hallucinators and 10% of the non-hallucinators showed increased muscle tension. These figures excluded cases where the presence or absence of hallucinations was unclear. Gould (1948) concluded:

> It is the writer's opinion that the activity of the vocal musculature is an essential factor in the production of a type of automatic speech which is more or less audible only to the patient (except under experimental conditions) and is misinterpreted as a "strange voice" (p. 371).

By means of auscultation of the larynx of a chronically hallucinated female schizophrenic patient, Gould (1949) was able to hear indistinct words. In order to investigate this patient's subvocal speech more fully, a sensitive microphone was placed close to her nose and mouth. Recordings were made of her voices on a plastic disc. In addition, electromyographic data were recorded from the lower lip. The recorded subvocal speech was roughly twice as fast as normal speech, differed on inspiration and expiration, and showed a different rhythm from the patient's external speech. There was a marked temporal correspondence between the recorded voices and the auditory hallucinations of the patient, as well as with activation of the electromyogram.

This work was refined on a series of 84 hallucinated patients (Gould, 1950) and the author proposed that the schizophrenic patients hear their own automatic speech. Gould hypothesized that the inner speech mechanism is activated during thinking and that during concentrated thought, whispering that is audible to the patient may be produced. This augmented subvocalization is typically produced without the patient's awareness and when it becomes conscious, it is misattributed to external sources.

Using a sophisticated psychophysiological assessment procedure McGuigan (1966) demonstrated increased respiration amplitude and chin muscle action potentials immediately preceding auditory hallucinations. This was consistent with the prediction of Gould (1950) that proprioceptive sensations should precede "hearing" voices. McGuigan also noted a tendency towards increased tongue muscle action potentials and whispered sound production just prior to reports of hallucinations. Control measures ruled out the possibility that these effects represented generalized body arousal and McGuigan concluded, that for at least one psychotic subject, increased covert oral language behavior preceded reports of auditory hallucinations.

The association between auditory hallucinations, inner speech, and electromyographic activity was further investigated by Inouye and Shimizu (1970). Nine medicated schizophrenic patients were studied, all of whom experienced complex auditory hallucinations. Stainless steel electrodes were inserted directly into the muscles of the lip and chin to record their electromyographic activity and a contact microphone was attached to the throat to record subvocal speech. Examination of the temporal relationship between onset of auditory hallucination and increase in EMG activity of the speech musculature showed that in almost half the cases they were related. The perceived loudness of the hallucination was also positively correlated with the degree of EMG activation. Hallucinations unaccompanied by EMG increase were perceived as quieter than those with EMG activation. The authors concluded that their data were generally consistent with the hypothesis that hallucinations are an expression of subvocal speech, a part of the inner speech mechanism.

These studies suggest that, for at least some schizophrenics, auditory hallucinations are produced by their own speech mechanisms. This issue was directly addressed by Green and Preston (1981), who were able to record and playback the subvocalization of a male schizophrenic patient in real-time. They noted that one of the effects of the amplified feedback was to raise the volume of the patient's "whispers" to a level where they could be heard as a form of distorted speech. The contents of the whispers, as rated by the experimenters, agreed with the patient's self-report of the auditory hallucinations. Although not establishing that the patient's subvocalizations were in fact his hallucinations, the authors point out that a close correspondence between the two would provide an empirical base from which to study hallucinations.

An elegant experiment by Bick and Kinsbourne (1987) concluded that subvocalization accompanies auditory hallucinations in schizophrenic patients and that schizophrenics generate the voices that they hear. Eighteen medicated and hallucinated psychiatric inpatients who met DSM III criteria for schizophrenia were included in the study. Subjects were asked to: (1) close their eyes tight, (2) open their mouth wide, and (3) clench their fists tightly. Each exercise was performed separately for one minute and the effect on the patient's hallucinations was recorded. Mouth opening selectively abolished the voices (13/18 cases), while clenching both fists removed the voices for 1 patient and closing the eyes removed the voices for 2 patients. For these same patients mouth opening also dispelled the voices. Bick and Kinsbourne also performed the same experiment on 21 normal controls who had been hypnotized into hearing voices. As with the schizophrenics mouth opening eliminated the voices (18/21 cases). They speculate as to the generality of this mechanism and whether it mediates voices resulting from temporal lobe lesions or those generated by direct stimulation of exposed cerebral cortex. It is interesting to note here that Shimizu and Inouye (1986) have reported a close association of phasic EMG discharges in speech muscles during dreaming with recall of speech in dreams.

Not all the evidence has supported the linkage between subvocalization and auditory hallucinations. Roberts, Greenblatt, and Soloman (1952) were unable to find consistent EMG activation in their group of 13 subjects. Of the 43 hallucinations reported by the 6 hallucinating subjects only 4 were linked to EMG bursts from the mouth and larynx. It was noted that resting levels of muscle tension were somewhat greater in the psychotic group than in the controls (N=3). More recently, Junginger and Rauscher (1987) in an attempt to replicate the work of Gould (1948) and McGuigan (1966) did find increased vocal EMG potentials in hallucinators. However, when a nonvocal muscle control (forearm extensor) was included in the analysis, the effect became non-significant. In addition no subvocalizations were detected in any subject during or following self-report of a hallucination. The authors cite evidence of increased covert speech muscle activity during normal verbal imagery and silent reading and they suggest that covert oral muscle activity may not be able to differentiate auditory hallucinations from normal verbal cognition.

Indirect support for the role of dysregulation of covert speech mechanisms, what Modell (1960) referred to as "hallucinated inner speech," comes from the work of Bazhin, Wasserman and Tonokonogh (1975). Employing a signal detection paradigm, they demonstrated an increase in right-ear threshold for detection of short-tone signals in hallucinated patients. In normal controls and in non-hallucinated patients, signal detection thresholds rose symmetrically as the signal tone shortened from 1000 to 1 millisecond. Results of this investigation were the same as previous work by this group on subjects with left temporal lobe pathology. The authors link their findings of left temporal abnormality in hallucinated psychiatric patients (dextrals only) to disturbances of inner speech systems. They also caution against attributing verbal hallucinations solely to cortical dysfunction of the temporal lobe, pointing out the importance of deep brain structures in hallucinations.

One consequence of the examination of subvocal speech in hallucinating patients has been the use of overt speech to block auditory hallucinations (Bick & Kinsbourne, 1987; Erickson & Gustafson, 1968; Green & Preston, 1981). It was found that use of the vocal chords, for example, humming or gargling (Erickson & Gustafson, 1968) naming objects aloud (Green & Preston, 1981), or even opening the mouth wide (Bick & Kinsbourne, 1987), all temporarily blocked auditory hallucinations. Thus overt speech appears to be incompatible with auditory hallucinations (i.e., covert speech). However, even if patients realize that they can quite easily stop their auditory hallucinations they are frequently unwilling to do so (Bick & Kinsbourne, 1987; Faloon & Talbot, 1981).

Overall, there is a significant body of evidence linking auditory hallucinations to abnormalities of the subvocal speech mechanism. In fact the work of Gould (1949, 1950), Green and Preston (1981), and Bick and Kinsbourne (1987) suggests that the subvocalization is the hallucination, a position also espoused by Flor-Henry (in press, a). Nevertheless not all findings are consistent with this view (Junginger & Rauscher, 1987; Roberts, Greenblatt & Solomon, 1952) and while it is agreed that increased EMG activation is associated with auditory hallucinations, not all auditory hallucinations will be matched by increased activation of the speech musculature (Inouye & Shimizu, 1970; McGuigan, 1966).

Abnormalities of language production mechanisms have been clearly linked to schizophrenia and to auditory hallucinations in particular (Hoffman, 1986). Therefore, it may be that dysregulation of the covert speech systems is only one component of a more generalized language dysfunction in schizophrenia. There does not appear to be a literature discussing the role of verbal dysregulation in organically induced hallucinations or in other forms of mental illness (e.g., manic-depressive illness, psychotic depression). In addition, methodological variables in electromyography have been refined over time (see Garrity, 1977, for a review of this topic) and early studies lack the sophistication of more recent work (cf. Gould, 1949; Junginger & Rauscher, 1987). Despite the conflicting evidence concerning the role of subvocal speech in the genesis of auditory hallucinations, it remains a fascinating and provocative observation that overt speech, that is talking aloud, will selectively abolish auditory hallucinations in schizophrenia.

Chapter IV

Method

4.1 Introduction

This chapter is divided into six separate sections: introduction, subject characteristics, design of the study, assessment, data collection procedures, and hypotheses. The section on subject characteristics describes the subjects in terms of numbers, age, sex, education, and handedness. In addition, subject selection and diagnostic criteria are presented. The design of the study is outlined in the third section. In the fourth section the procedures used in the investigation are discussed. Section five describes neuropsychological tests used in the collection of data for the dissertation, and the final section of the methodology chapter presents the hypotheses that are tested.

4.2 Subject Characteristics

Three groups of subjects provide the data for this investigation. They are: a group of selected psychiatric inpatients, with and without hallucinations, who satisfied research diagnostic criteria for Mania, Schizophrenia, and Endogenous Depression; a group of heterogeneous hospitalized inpatients, with and without hallucinations, who were unselected for diagnoses, and a normal control group. Age, sex, handedness, IQ, and educational data are reported for each group in Table 5.1.

4.2.1 Selected Psychiatric Inpatients

The group of selected psychiatric inpatients consisted of 54 subjects (20 hallucinators, 34 non-hallucinators). Criteria for inclusion in the group was that of Taylor and Abrams (1978, pp. 945-946) and Taylor, Abrams, and Gaztanaga (1975, pp. 91-92). These are summarized as follows:

A. Schizophrenia (all of 1 through 4):
  1. At least one of (a) through (c):
    (a) formal thought disorder (driveling, tangentiality, neologisms, non sequiturs, private words, paraphasias, stock words);
    (b) first rank symptoms (at least one); and
    (c) emotional blunting (a constricted, inappropriate, unrelated affect of decreased intensity, with indifference/unconcern for loved ones, lack of emotional responsivity, and a loss of social graces).
  2. Clear consciousness.
  3. No diagnosable affective disorder.
  4. No diagnosable coarse brain disease, no past hallucinogenic or psychostimulant drug abuse, and no medical condition known to cause schizophrenic symptoms.

B. Mania (all of 1 through 4);
  1. Hyperactivity.
  2. Rapid/pressured speech.
  3. Euphoric/expansive/irritable mood, with a broad

affect.

4. No diagnosable coarse brain disease, no psychostimulant drug abuse in the past month, and no medical illness known to cause manic symptoms.

C. Endogenous Depression (all of 1 through 3);

1. Sad, dysphoric, or anxious mood.
2. Three of (a) through (f):
   - (a) early morning wakening,
   - (b) diurnal mood swing (worse in a.m.),
   - (c) weight loss of more than five pounds in three weeks,
   - (d) retardation or agitation,
   - (e) suicidal thoughts/behavior, and
   - (f) feelings of guilt/hopelessness/worthlessness.
3. No diagnosable coarse brain disease, no use of steroids or reserpine in the past month, and no medical illness known to cause depressive symptoms.

4.2.2 Unselected Psychiatric Inpatients

This group was unselected in terms of psychiatric diagnosis and consisted of 107 hospitalized patients, 55 of whom experienced hallucinations as part of their current symptomatology. These cases were referred for assessment to the Department of Neuropsychology at Alberta Hospital Edmonton and consisted primarily of diagnostic and treatment problems.

4.2.3 Normal Control Group

Control data consist of 182 normal volunteers (104 males, 78 females) with no reported history of psychiatric or neurological illness. Exclusion criteria included any of the following: significant bleeding, toxicity or other complications during their mother's pregnancy, birth complications (e.g., anoxia, use of forceps causing significant contusions or hematomas), low birth weight, febrile convulsion, encephalitis or epilepsy, meningitis, closed head injuries associated with postconcussional sequelae, neurological disorders of the central nervous system, psychiatric illness, or an alcoholic history (Fromm-Auch & Yeudall, 1983; Yeudall, Fromm, Reddon, & Stefanyk, 1986; Yeudall, Reddon, Gill, & Stefanyk 1987).

4.2.4 Assessment of Hallucinations

For the selected sample (N=54) the presence of hallucinations, as part of the clinical presentation, was established by retrospective examination of the case notes by two psychiatrists who also established the patient's diagnosis, using Taylor and Abrams (1978), and Taylor, Abrams, and Gaztanaga (1975) criteria. These subjects were unmedicated dextrals as defined by the hand used for writing (Annett, 1970). The unselected patients (N=107) were diagnosed by their attending physician and the presence of hallucinations was established through case review by the author, or by means of interview at case conference. Where possible unmedicated patients were tested, unless of course ethical or clinical requirements necessitated pharmacological intervention. Both groups of inpatients were tested early in their admission to hospital. Only subjects with essentially complete data are included in the investigation.

4.3 Design of the Dissertation

This investigation uses a case-control design and is comprised of two studies. The first study consists of an evaluation of selected psychotic patients with and without hallucinations and normal control subjects. The second study consists of an evaluation of heterogeneous psychotic patients with hallucinations and normal control subjects. Additionally, comparisons will be made between selected and unselected hallucinators. In each study summary statistics will be age and sex equated with the recently published norms of Yeudall, et al. (1986) and Yeudall, et al., (1987) to produce norm referenced profiles. Composite (group average) neuropsychological profiles will be generated for normal controls, hallucinated psychotic patients and non-hallucinated psychotic patients. These groups will be compared by analysis of variance procedures to determine differences between controls and selected hallucinators, controls and unselected hallucinators and selected hallucinators and unselected hallucinators. In the event of significant overall group differences, univariate follow-up will be conducted to determine variables upon which the groups differ.

Questions investigated include: (1) do the groups differ? and (2) how do they differ (i.e., which group is more impaired)? If a pattern of group differences, exists a neuropsychological model of hallucinations will be developed.

A second group of hospitalized inpatients will be analyzed by similar procedures, resulting in a norm referenced composite profile of heterogeneous hallucinators. This group of hallucinators will be compared to the derivation sample of psychotic hallucinated patients and psychotic non-hallucinated patients as well as to normal controls. In the event of significant overall group differences, post-hoc univariate follow-up will be conducted to determine variables upon which the groups differ.

The first group of hallucinated patients reflects relatively pure forms of three specific diagnostic groups that are characterized by hallucinations: Mania, Depression, and Schizophrenia (Taylor & Abrams, 1978; Taylor, Abrams, & Gaztanaga, 1975). The second group is heterogeneous diagnostically and was collected on the basis of presence or absence of the symptom of hallucinations (Persons, 1986). It is therefore more representative of the cases that flow through the admission wards of psychiatric hospitals.

4.4 Procedures

This section summarizes the test setting, the background of technicians administering the test battery, and the basic principles of interpretation.

4.4.1 Test Setting

All subjects were assessed in testing laboratories utilized by the Department of Neuropsychology at Alberta Hospital Edmonton. The test laboratories are designed to be distraction free and they contain a Halstead-Reitan neuropsychological test battery, as well as additional tests employed by the department. Subjects are seated across a table from the technician who administers the battery. For subjects judged to be unpredictable, a laboratory with an emergency buzzer system is used. Testing is voluntary and authorized by the referring physician, to whom the results of the assessment are communicated. The time taken to complete the test battery averages between five to eight hours, with breaks taken to minimize subject fatigue.

4.4.2 Technicians

The Department of Neuropsychology provides comprehensive training in the administration, scoring, and computer entry of an expanded Halstead-Reitan neuropsychological test battery. This battery was originally developed in the late 1960's by the director of the Department of Neuropsychology and it has been validated on both normal and brain damaged subjects (Aftanas & Royce, 1969; Royce, Yeudall, & Bock, 1976). Technicians employed by the department are taught to administer the battery by a neuropsychologist who is also a chartered psychologist in the Province of Alberta.

Following training, which currently involves videotaping and feedback from experienced technicians, initial patient assessments are supervised by a neuropsychologist observing from behind a one-way mirror.

In addition to on the job training provided by the Department of Neuropsychology, technicians also have either a Bachelor's degree in Psychology or two years of training in Behavioral Sciences from a post-secondary institution. The technicians are taught test instructions verbatim and test administration order is fixed for specific test batteries. These restriction may be relaxed in order to ensure subject compliance and also that the subject comprehends the test instructions. A computerized system of validity vectors has been developed in order to alert the interpreting clinician to any changes in test standardization.

4.4.3 Interpretation of Test Data

Following administration, scoring, and computer entry of the test data, the results are interpreted by a clinician who is supervised by the department director. Clinicians have a minimum educational level of a Master's degree in Psychology, with relevant course work. Interpretation is based on the methods developed by Reitan (1959; 1967; 1974) and Selz and Reitan (1979). These include evaluation of: (a) level of performance, (b) pathognomonic signs, (c) pattern of performance, and (d) comparison of the two sides of the body. Additionally, results are examined to determine qualitative patterns of performance and compared to available indices of premorbid functioning.

4.5 Materials: Neuropsychological Test Battery

Assessment of cognitive, motor and sensory-perceptual abilities is accomplished by the use of the Wechsler Adult Intelligence Scales and a battery of fifteen neuropsychological tests. Full details of administration and scoring for the WAIS (1955) and WAIS-R (1981) can be found in their respective manuals and additional descriptions of the subtests are to be found in Matarazzo (1972), Matarazzo and Matarazzo (1984), and Zimmerman and Woo-Sam (1973).

The neuropsychological tests used in the investigation are listed in Table 4.1 and represent a modified and expanded Halstead-Reitan battery. Description of the Halstead Category Test, Seashore Rhythm Test, Speech Sounds Perception Test, Finger Tapping Test, Trailing Making Test, and Hand Dynamometer can be found in Boll (1981); Golden, Osmon, Moses, and Berg (1981); Reitan (1966, 1969); Reitan and Davison (1974); Reitan and Heineman (1968); Reitan and Wolfson (1985); Trites (1981), and Swiercinsky (1978). The preceding measures are well established in the literature and need not be further elaborated upon.

Table 4.1

Scoring Metrics for Neuropsychological Variables

VARIABLE	UNIT OF SCORING
Colored Progressive Matrices	Total number of errors (CPM)
Oral Word Fluency	Mean number of words per letter in 60 seconds (OWF)
Purdue Pegboard	Number of pegs placed (PURDP, PURDNP, PURDB) Number of assemblies constructed (PURDA)
L.T. Tactile Recognition	Time in seconds (LJPT, LJNPT, LJBT)
Wisconsin Card Sorting Test	Total number of errors (WCST) Number of categories sorted (WCSCAT)
Speech Sounds Perception Test	Total errors (SPEECHS)
Trail Making Test	Time in seconds (TRAILA, TRAILB)
Halstead Category Test	Total number of errors (HALCAT)
Finger Tapping Test	Mean number of taps in five trials (FTAPP, FTAPNP)
Dynamometer	Kilograms (DYNP, DYNNP)
Seashore Rhythm Test	Total number of errors (SEAR)
Finger Localization	Total number of errors per condition (FLOCPSS, FLOCPDSS, FLOCNPSS, FLOCNPDS)

4.5.1 Non Halstead-Reitan Neuropsychological Tests

Information on the administration, validity, and reliability of the Raven's Colored Progressive Matrices, Finger Localization Test, Oral Word Fluency Test, Purdue Pegboard, L.J. Tactile Recognition, and Wisconsin Card Sorting Test is available in Fromm-Auch and Yeudall (1983); Lezak (1983); Lishman (1978, 1987); Strub and Black (1985), and Yeudall, Fromm, Reddon and Stefanyk (1986). Because these tests are less well known than the Halstead-Reitan measures, they will be reviewed below.

Tests are discussed in terms of (a) a general description, what they measure and how they are scored, (b) psychometric properties, (c) subject groups that they have been utilized with, and (d) neuropsychological implications of the test. Where psychometric studies are lacking, sections (b) and (c) are placed together. Instructions for the administration of these tests are presented in Appendix III.

4.5.1.1 Colored Progressive Matrices

General Description: Developed as a "culture fair" measure of perceptually mediated intellectual ability in children aged 5 to 11 years and adults of 60 and over, (Raven, 1949; Raven, 1965; Raven, Court, & Raven, 1977) the test consists of 36 items organized into 3 subtests. Printed on brightly colored paper, items are presented in a horizontal format and require the subject to identify the correct shape necessary to complete a design or pattern. As the subject moves through the three subtests, there is an increase in the cognitive complexity of the task. The test, which is untimed, begins with gestalt completion items and progresses to simple analogies. It requires visuospatial analysis, sustained attention, and the ability to cope with unfamiliar visual patterns (Costa, 1976; Lezak, 1983).

Psychometric Properties: The test has been shown to be reliable for children and not to show sex or race effects. Carlson and Jensen (1981) report test-retest reliability coefficients ranging from .81 to .87 in a large sample of 783 children between the ages of 5 1/2 years and 8 1/2 years. These values are consistent with those reported by Raven (1965). A split-half reliability coefficient of .896 is reported by Freyberg (1966) on a sample of 159 children aged 6 to 7 years. Schmidtke and Schaller (1980) report a 3 factor solution for the test accounting for 38.6% of the total variance (principal components with varimax rotation). The factors identified were, (a) perceptual closure involving complex figures and patterns with heterogeneous inner structures, (b) concrete and abstract reasoning, and, (c) completion of homogeneous patterns and recognition of given elements. The authors report their findings to be consistent with previous factor analytic studies of the Colored Progressive Matrices.

Age effects have been demonstrated for adults by

Panek and Stoner (1980), who found more errors in the performance of older adults. Mean scores are comparable to those reported by Raven (1965) for older, healthy adults. A computerized version of the test has been developed and preliminary work shows it to be similar to the traditional form, in terms of the total error score (Rock & Nolen, 1982).

Subject Groups: In the field of neuropsychology, the Coloured Progressive Matrices test has been found to be effective in the discrimination of brain damaged from control subjects. Dils (1960) correctly identified 82% of organically impaired subjects and 92% of control subjects. The two groups of 60 subjects were matched for age, sex, race, and education. Evans and Marmorston (1964), utilizing four different scoring systems, were able to correctly classify 77% of brain damaged and 60% of non-brain damaged subjects. Raven's raw score proved to be the most effective of the four systems. One possible reason for the lower correct classification rates in this study was the use of control subjects who had all recovered from acute myocardial infarctions. Thus some controls may also have had brain damage or impairment.

Neuropsychological Implications: In terms of cerebral localization, the test is thought to be most sensitive to right hemisphere damage, particularly post-Rolandic lesions (Costa, 1976; Denes, Semenza, Stoppa, & Gradenigo, 1978; Gainotti, Caltagirone, & Miceli, 1977). This effect has been investigated and shown to be due to spatial neglect in right brain damaged subjects. A comprehensive analysis of this issue by Gainotti, D'Erme, Villa, and Caltagirone (1986) demonstrated that if the effects of age, education, lesion size, and unilateral spatial neglect are controlled, then no significant differences between left and right brain damaged subjects are observed.

These authors also showed that within the left brain damaged group, aphasics with severe language comprehension disorders scored worse than non-aphasics. Results are based on a vertically presented, "neglect free" modification of the original test format. They conclude that for clinical practice, the regular version of the Colored Matrices test is preferred, and that the modified test is a "research version" to be used for the specific assessment of "...the intellectual ability of brain-damaged patients partialling out the influence of unilateral spatial inattention" (Gainotti, D'Erme, Villa, & Caltagirone, 1986, p. 49).

4.5.1.2 Finger Localization Test

General Description: Modified from the procedures of Reitan's Finger Agnosia Test (Reitan, 1969) and Benton's Finger Localization procedures (Benton 1955; 1959), this sensory-perceptual measure requires subjects to identify, without visual guidance, fingers touched by the examiner. Starting each of the 11 subtests with the preferred hand, verbal and nonverbal responses are elicited on trails of both single and double sequential stimulation. Depending on the subtest, the subject is asked to identify the finger or fingers stimulated by either: (a) pointing, (b) naming, (c) indicating the finger on the hand of the examiner, or (d) indicating the number of the finger on a drawing. A total error score summed across all subtests is obtained for each hand. In addition, separate scores for single and double errors are recorded.

Psychometric Properties/Subject Groups: Normative data for this test is reported by Fromm-Auch and Yeudall (1983) and Yeudall, et al. (1987). For Benton's original method, normative data are reported in Benton, Hamsher, Varney, and Spreen (1983). Psychometric studies of this tests are generally lacking and each group of investigators will typically modify the test to suit its own needs (e.g., Gainotti, Cianchetti, & Tiacci, 1972 substituted pointing for verbal responses in order to minimize aphasic bias).

Neuropsychological Implications: Finger localization deficits may be produced by lesions of either hemisphere (Lezak, 1983), consequently, interpretation of errors on this task is complex. Bilateral impairment has been shown to be associated with aphasia and/or generalized mental impairment (Benton, 1979; Benton, Hamsher, Varney, & Spreen, 1983). Both diffuse brain injury and dominant parietal lesions have been linked to errors on this test

(Strub & Black, 1985). Reitan and Wolfson (1985) state that posterior cerebral lesions are primarily responsible for finger agnosia deficits. The linkage of finger localization and the dominant hemisphere (Benton, Hammer, Salzinger, & Sutton, 1972) appears to be a function of the presence of aphasia (Poeck & Orgass, 1969).

Traditionally, finger localization has been intimately associated with Gerstmann's syndrome, a select group of deficits that includes finger agnosia, right-left disorientation, agraphia, and acalculia. The work of Benton (1959; 1961; 1977), Ettlinger (1963), and Kinsbourne & Warrington (1962) has shown, however, that measures of finger localization are multifactorial in nature and that level of performance alone has little localizing value.

4.5.1.3 Oral Word Fluency

General Description: Also known as the Verbal Associative Fluency test (Benton, 1968) and later as the Controlled Word Association test (Benton & Hamsher, 1976), this measure of verbal fluency requires the subject to say as many words as possible, within a set time limit, that begin with a specified letter. The version in this study utilizes the letters F, A, and S. The verbal associative frequency of these letters was determined by Borkowski, Benton, and Spreen (1967), who found that controls produced, on average, 10 or 11 words for each of the letters F, A, and S. Subjects are asked to say as many words as possible in one minute, but they may not use proper nouns, numbers, or variations of the same word with different endings (Borkowski, Benton, & Spreen, 1967).

In order to perform successfully on this test, subject's must organize their thinking, restrict their output within the limits imposed by the test instructions, and recall which words they have already used (Estes, 1974).

Psychometric Properties/Subject Groups: The test is influenced by age, sex, premorbid verbal skills and years of education (Berg, Franzen, & Wedding, 1987; Lezak, 1983), although Benton, Eslinger, and Damasia,(1981) did not find the expected decline with age in their sample of 65 to 84 years old control subjects. Rosen (1980) also found the controlled word association test to be resistant to the effects of aging in the elderly.

Neuropsychological Implications: Impaired verbal associative fluency has been associated with brain damage (Milner, 1967, Strub & Black, 1985) and is especially impaired by the presence of aphasic deficits or frontal lobe damage (Benton, 1968; Lezak, 1983). In particular, dominant frontal injury, anterior to Broca's area has been implicated (Milner, 1971; Perret, 1974; Taylor, 1979).

It was noted by Benton (1968) and more recently by Miceli, Caltagirone, Gainotti, Masullo, and Silveri (1981) that right frontal and bifrontal lobe damage can also impair performance on this test. Within a neuropsychiatric sample Crockett, Bilsker, Hurwitz, and Kozak (1986) found word fluency to be sensitive to frontal lobe pathology, while Rosen (1980) reports a selective impairment of verbal associative fluency in elderly subjects with mild senile dementia Alzheimer type.

4.5.1.4 Purdue Pegboard

General Description: This psychomotor test of manual dexterity was developed in the 1940's by the Purdue Research Foundation (1948) as a measure for employment selection. It requires fine motor skill, finger dexterity, visual discrimination skills, and responsiveness to visual and tactile feedback (Bourassa & Guion, 1959; Fleishman & Ellison, 1962; Fleishman & Hempel, 1954). Subjects place metal pins in a specially constructed board using their preferred, nonpreferred, and both hands. Each trial, which is preceded by practice, lasts 30 seconds. Additionally, subjects are required to construct assemblies consisting of a pin, washer, collar, and second washer. This trial lasts 60 seconds and speed is emphasized throughout the test, which may be given once or repeated three times for each section. The score is the number of pegs placed in each trial (right, left and both hands) and the number of parts assembled in the construction trial (Tiffin, 1968).

Psychometric Properties: Considerable normative data, across a wide range of different groups, has been collected for this test. For children and adolescent subjects see: Costa, Scarola, & Rapin, 1964; Gardner &

Broman, 1979; Mathiowetz, Rogers, Dowe-Keval, Donahoe, & Rennells, 1986; Wilson, Iacoviello, Wilson, & Risucci, 1982). For adult norms (see, Hamm & Curtis, 1980; Tiffin, 1968; Tiffen & Asher, 1948). The test is sensitive to sex, with females scoring slightly higher than males (Lezak, 1983; Mathiowetz, Rogers, Dowe-Keval, Donahoe, & Rennells, 1986; Sattler & Engelhardt, 1982; Yeudall, et al. 1986). Age, but not education effects have been reported for older subjects (Costa, Vaughan, Levita, & Farber, 1963; Hamm & Curtis, 1980).

Studies of the reliability of this test are encouraging. Tiffin and Asher (1948) report single trial test-retest reliability coefficients of .63 to .68 on a sample of college students. Tiffin (1968) reports single trial test-retest correlations ranging from .60 to .76 in the Purdue Pegboard manual. It is noted that the three trial procedure is more reliable, with coefficients ranging from .82 to .91. Using a sample of 400 basic airmen, Fleishman and Hempel (1954) report single trial test-retest reliability coefficients of .68 to .74. On immediate retesting with a sample of 104 children, Wilson, Iacoviello, Wilson, and Risucci (1982) report test-retest reliability coefficients of .85 to .92.

Recently, Reddon, Gill, Gauk and Maerz (1988) reported on the temporal stability of the Purdue Pegboard over 5 repeated administrations (once per week, for 4 weeks) with normal subjects. For the single trial administration, they report mean test-retest reliability coefficients for males/females of .63/.76 for the right-hand, .64/.79 for the left-hand, .67/.81 for both-hands, and .81/.83 for assemblies. The same statistic for the right/left hand ratio was .33 for males and .22 for females, somewhat lower than the single test-retest ratios of .61 and .48 reported by Sappington (1980).

Subject Groups: Different subject groups have been investigated with the Purdue Pegboard. Serafetinides and Clark (1973) found impairment on the Purdue following single dose administration of antipsychotic medications, in a sample of drug free schizophrenics. Fromm and Schopflocher (1984) reported no change in pegboard scores following a trial of antidepressants, in a sample of hospitalized psychiatric patients. Mack (1969) found the Purdue Pegboard to be ineffective in discriminating brain damaged from non-brain damaged psychiatric patients. Brain damage was diagnosed primarily on the basis of a physical examination, rather than a complete neurological assessment. Impairment on this measure has also been reported for learning disabled children (Kane & Gill, 1972; Leslie, Davidson, & Batey, 1985).

Neuropsychological Implications: As a screening tool for the assessment of brain damage, the Purdue Pegboard has shown considerable utility. Costa, Vaughan, Levita, and Farber (1963) report correct classification of 89% of brain damaged subjects and 70% accuracy in classifying the laterality of lesions. This shrunk to 60% upon cross-validation, but the overall correct identification of brain damaged subjects remained at 89%. Vega (1969) correctly identified 80% of a small sample of brain damaged subjects and 85% upon cross-validation. Golden (1979) reports overall accuracy averages of about 80% for the Purdue Pegboard.

Working with children suffering from organic brain disorders Rapin, Tourk, and Costa (1966) correctly classified 78% of their subjects. They also found the test to be useful in predicting laterality of lesion, but it was insensitive to chronic epilepsy that was without associated mental retardation. Increased sensitivity to right hemisphere damage is reported by Vaughan and Costa (1962) and in their factor analysis of brain damaged subjects, Royce, Yeudall, and Bock (1976) found the Purdue Pegboard to be sensitive to right frontal and right parietal damage.

4.5.1.5 L.J. Tactile Recognition

General Description: This task was designed in 1972 by L. Yeudall and J. Kisilevich to be a nonverbal equivalent to the Tactile Form Recognition test used in the Halstead-Reitan battery. The task emphasizes nonverbal mediation due to the asymmetric nature of the stimulus shapes (i.e., they are difficult to encode verbally, whereas the circle, square, triangle, and cross used in the Tactile Form Recognition Test are easy to code verbally). Instructions for this test are based on those for the Tactile Form Recognition test (Reitan & Wolfson, 1985). Without visual guidance the subject is given one asymmetrical shape at a time to feel and is required to identify it, by pointing, from an array of four shapes. The times taken to point to the appropriate shapes in the array and the number of errors for the preferred, nonpreferred, and both hands are recorded.

Psychometric Properties/Subject Groups: Inspiration for the task came from the tactile nonsense patterns used by Milner (1971) who reported right hemisphere superiority for the perception of spatial patterns. She also noted that the nonsense shapes were chosen because they did not easily lend themselves to verbal coding and were therefore useful right hemisphere measures. Lezak (1983) cites evidence that nonverbal configured tasks are more difficult than their verbal counter parts.

Neuropsychological Implications: Tests of sensory-perceptual functioning are mediated primarily by the parietal lobes (Bigler, 1987; Gilandas, Touyz, Beumont, & Greenberg, 1984; Golden, 1979; Golden, Osmon, Moses & Berg, 1981) and may show a right hemisphere bias (Walsh, 1978). The L.J. Tactile Recognition task emphasizes nonverbal mediation and because of this is intended to be more sensitive to right parietal functions than the Tactile Form Recognition test. The normative data of Yeudall, et al. (1986) report no age or sex effects for errors scores, but some age effects for the nonpreferred and both hand time scores. Normal subjects make very few errors on this test.

4.5.1.6 Wisconsin Card Sorting Test

General Description: Developed as an objective and quantifiable measure of, "human abstraction and shift of set" (Berg, 1948; Grant & Berg, 1948) this test has gained wide clinical acceptance (Lezak, 1983). In the present investigation, the Wisconsin Card Sorting Test (WCST) manual and cards as distributed by Wells Printing Company were used (Grant & Berg, undated). A slightly modified version has been developed by Heaton (1981) and is distributed by Psychological Assessment Resources.

Subjects are provided with a deck of 64 cards on which one to four symbols are printed. These include a star, cross, triangle, or circle and stimuli may be red, green, yellow, or blue in color. None of the cards are identical. The subject must sort the cards based on the principle of color, form, or number. The examiner may only say "right" or "wrong" in response to the subject's sorting strategy. Shifts occur, without warning, after ten correct responses. Discontinuation criteria are six categories of ten cards each (color, form, number, color, form, number) or use of more than sixty-four response cards, without completing a single category of ten consecutive correct responses. Heaton (1981) reviews different criteria that researchers have used to administer and score the WCST.

The WCST measures an array of skills, including the ability to form concepts, think flexibly, respond to external feedback, and modify or maintain a mental set (Heaton, 1981; Lezak, 1983; Tarter & Parsons, 1971). Crucial to effective performance on the WCST are the abilities to form concepts, shift in response to feedback, and maintain the relevant set in spite of the shifts. Additionally, Stuss and Benson (1984, p.18) comment that patients may display, "a dissociation between knowing and doing." That is, they can verbalize the concepts of color, form, and number, but are unable to use this knowledge to modify ongoing behavior.

Psychometric Properties/Subject Groups: The WCST has been employed with a variety of psychopathological and neurological populations. These include: alcoholics (Klisz & Parsons, 1979; Tarter, 1973), epileptics (McDaniel & McDaniel, 1976), autistic patients (Rumsey, 1985), psychopaths (Hare, 1984), Huntington's disease (Josiassen, Curry, Mancall, Shagass, & Roemer, 1986), Parkinson's disease (Bowen, Kamienny, Burns, & Yahr, 1975), Multiple Sclerosis (Rao, Hammeke, & Speech, 1987), schizophrenics (Fey, 1951; Kashima et al., 1985; Kolb & Wishaw, 1983; Malmo, 1974), and mixed neuropsychiatric patients (Crockett, Bilsker, Hurwitz, & Kozak, 1986). In addition, the WCST has been used with neurologically impaired children (Chelune & Thompson, 1986) and children's norms are available (Chelune & Baer, 1986).

There are several modifications of the original WCST procedures, the best known being those of Heaton (1981) and Nelson (1976). A computerized version is also available (Beaumont, 1981), and Weinberger, Berman, and Zec (1986) used an automated form of the WCST in their regional cerebral blood flow study of schizophrenics.

While little information is available on the psychometric properties of this test, age effects have been reported for older subjects (Loranger & Misiak, 1960; Nelson, 1976). Heaton (1981) finds education significantly correlated with WCST performance in normals, but not in brain damaged subjects. Several studies have compared the WCST with the Halstead Category Test (HCT) which also requires abstract reasoning and conceptual flexibility. The most consistent finding from these studies is that while the two tests are related, they are not measuring the same thing, and are therefore not interchangeable (Bond & Buchtel, 1984; Brandon & Chavez, 1984; King & Snow, 1981; Pendleton & Heaton, 1982; Perrine, 1986; Rothke, 1986).

Subject Groups: As a measure of cerebral functioning the WCST has shown a documented sensitivity to frontal lobe damage, particularly in the prefrontal region. The original work is that of Milner (1963, 1964) who found that frontal lobe lesions invading the dorsolateral convexity produced an inability to shift sorting principles on the WCST. Orbitofrontal lesions had no such effect, neither did temporal lobe lesions, and in the early postoperative period no laterality effects were observed. While it is generally agreed that the WCST is sensitive to frontal or prefrontal lesions, its association with left frontal damage has been questioned. Milner (1971, 1975) suggested that left frontal structures were especially crucial in completion of this test. Malmo (1974), Drewe (1974), and Taylor (1969) confirmed these findings, however, Robinson, Heaton, Lehman, and Stilson (1980) and Bornstein (1986) found no laterality effects, although both of these studies agreed as to the sensitivity of the WCST to frontal lobe lesions.

An early study by Teuber, Battersby, and Bender (1951) did not find the WCST specifically sensitive to frontal lobe lesions. These authors used a modified card sorting test with forced shifts every ten trials, regardless of the patient's response set before it was shifted (Walsh, 1978). A recent cerebral blood flow study by Weinberger, Berman, and Zec (1986), using normal and schizophrenic subjects, found a failure to activate the dorsolateral prefrontal cortex in schizophrenics performing the WCST. Normals showed increased flow in the dorsolateral prefrontal cortex and performed substantially better on the WCST than the schizophrenics.

The most specific localization for the WCST is found in Kolb and Wishaw (1985), who state that while affected by both left and right frontal lesions, the principle site of effect for the WCST is around Brodman's area 9, in the left hemisphere. This is interesting in view of the report of Zangwill (1966) who summarizes Kleist's (1934) view that injuries to the posterior portion of the second frontal convolution of the left hemisphere, i.e., Brodman's area 9, is related to defects in verbal thought processes.

4.6 Hypotheses to be Tested

Placed in the null hypothesis form, the following hypotheses are to be tested:

1. There are no differences between psychiatric patients and controls, as assessed by a battery of neuropsychological tests.
2. There are no differences between unselected non-hallucinating patients, unselected hallucinating patients, selected non-hallucinating patients, and selected hallucinating patients, as assessed by a battery of neuropsychological tests.
3. There are no differences between unselected and selected psychiatric patients, as assessed by a battery of neuropsychological tests.
4. There are no differences between unselected non-hallucinating patients and selected non-hallucinating patients, as assessed by a battery of neuropsychological tests.
5. There are no differences between unselected

hallucinating patients and selected hallucinating patients, as assessed by a battery of neuropsychological tests.

6. There are no differences between unselected non-hallucinating patients and unselected hallucinating patients, as assessed by a battery of neuropsychological tests.
7. There are no differences between selected non-hallucinating patients and selected hallucinating patients, as assessed by a battery of neuropsychological tests.

The term "selected" refers to those patients who are diagnosed according to the research diagnostic criteria of Taylor and Abrams (1978) and Taylor, Abrams, and Gaztanaga (1975). The term "unselected" refers to those patients who have been assessed/diagnosed as part of the normal flow of hospital admissions.

Chapter V

Results

5.1 Introduction

This chapter consists of four sections. Firstly, methods of data screening prior to analysis are presented, followed by summary statistics for all groups. Then results of multivariate analysis of variance are reported, and the last section presents supplementary analyses of the data.

5.2 Data Screening Procedures

Five groups of patients were investigated: unselected non-hallucinators, unselected hallucinators, selected non-hallucinators, selected hallucinators, and controls. Unselected patients were individuals referred for neuropsychological assessment, irrespective of clinical diagnosis, and who did or did not have hallucinations as part of their presenting symptoms. Selected patients were those cases referred for neuropsychological assessment who met strict diagnostic criteria for Mania, Schizophrenia, or Psychotic Depression, and who did or did not have hallucinations as part of their clinical picture.

A listing of all tests by groups was prepared and examined for missing data. Fifteen tests were selected on an initial sample of 431 subjects. Raw scores for these tests were generated by group and by sex. This data was examined manually for unusual or extreme scores. When found, raw data was rechecked for accuracy or completeness. Values that were extreme, but representative of a subject's actual performance were included in the analysis. Clerical or data entry errors were corrected.

In addition the age distributions for all five groups were examined. In order to maintain an adequate number of subjects in each age band the age range in the study was restricted to 15 to 30 years and 31 to 45 years inclusive so that two age groups could be used. This resulted in a subject pool of 393. Examination of the population from which subjects for this study were drawn (N=4,208) showed that 78% of males and 77% of females were in the 15 to 45 year age range. Consequently, subjects are typical.

Fifteen tests were selected for analysis and all groups again screened for missing data. Subjects with more than five out of thirty five variables missing were excluded from the analysis. This reduced the subject pool to 343 (161 patients, 182 controls). Missing data was generated by a rank estimation procedure in which a subject's average rank on their good data was obtained and used in place of missing data. Where necessary, test scores were reflected so that all variables were in the same direction. Missing data was estimated on cases with 5 or fewer missing variables out of 35 possible variables. The justification for using ranks is discussed in section 5.3 below.

In order to facilitate data analysis, Wechsler Adult Intelligence Scale (WAIS) scores were converted to

Wechsler Adult Intelligence Scale-Revised (WAIS-R) scores by means of a linear equating procedure (Jackson, 1984). Initial equating was at the subtest level, and used the standard linear equating formulas of Angoff (1971). The second stage produced Verbal, Performance, and Full Scale IQ scores. This was done by summing scaled scores and entering the appropriate age group referenced IQ tables in the WAIS-R manual (Wechsler, 1981). The equating process assumes that the only differences between the distributions of two tests are the mean and variance and that both measures assess the same trait with equal reliability (Crocker & Algina, 1986).

In summary, data for analysis on five groups was prepared. These groups included unselected non-hallucinators, unselected hallucinators, selected non-hallucinators, selected hallucinators, and controls. Of the initial sample of 431 subjects, 343 were accepted for further statistical analysis. Subjects missing more than 5 out of 35 variables were eliminated. Of the fifteen neuropsychological tests initially chosen, three were discarded due to incomplete data. The subject pool was restricted to ages 15 to 45 years. This age band is representative of the population from which the data were drawn. WAIS scores were converted to the WAIS-R metric by means of linear equating, and missing data generated by means of a rank estimation procedure.

5.3 Summary Statistics

The sample consisted of 343 subjects (197 males, 146 females) with a mean age of 28.4 years (SD=7.3 years). Of these 182 were controls and 161 hospitalized psychiatric inpatients. Demographic characteristics for each of the five groups are reported in Table 5.1. These include sample size, mean age, sex, mean Verbal, Performance, and Full Scale IQ, level of education, and handedness. Scoring metrics for the neuropsychological tests used in the investigation are presented in Table 4.1. Tables 5.2 to 5.6 give summary statistics for each of the five groups by variable. A Chi Square test of independence showed no effects for group membership by sex, handedness by group, or age by sex. Due to the problem of small expected cell counts (Haeberman, 1988) Chi-Square data are not reported for vocational or academic education by group, sex, or age.

Because of distributional issues concerning neuropsychological test data, not only means and standard deviations are reported, but also medians, ranges, skewness and kurtosis (Lezak, 1984; Russell, 1987). In addition, Table 5.7 reports two tailed p values for the Kolmogorov-Smirnov Goodness of Fit test for a normal distribution. This measure is designed for use with ordinal data and is regarded as having more power than the Chi-Square test (Conover, 1980; Harnett, 1982). It can been seen from this table that many of the variables used in this investigation are not normally distributed and

Table 5.1
Demographic Characteristics of All Subject Groups

	Group 1	Group 2	Group 3	Group 4	Group 5
Sample Size	52	55	34	20	182
Mean Age (Range)	28.4(7.9) (16-44)	27.9(7.5) (16-44)	29.5(7.0) (19-45)	31.5(7.2) (19-43)	24.8(6.9) (15-45)
Sex:					
Males	32(61.5)	33(60.0)	18(59.9)	10(50.0)	104(57.0)
Females	20(38.5)	22(40.0)	16(47.1)	10(50.0)	78(43.0)
Mean WAIS-R					
VIQ	98.7(14.5)	88.4(13.8)	92.6(16.0)	88.0(13.3)	112.7(10.4)
PIQ	96.6(13.8)	87.3(13.4)	84.7(14.6)	82.5(10.9)	108.9(10.3)
FSIQ	97.5(13.2)	86.9(13.2)	88.4(14.8)	85.2(11.1)	111.9(9.5)
Education:					
Academic	11.5(2.9)	11.1(2.3)	12.1(2.7)	10.8(2.5)	13.9(2.5)
Vocational	0.4(1.0)	0.3(0.8)	0.3(0.9)	0.2(0.4)	0.2(0.9)
Handedness: (Writing Hand)					
Right	48	46	30	19	143
Left	04	08	04	01	21

Group 1: Unselected Non-Hallucinators
Group 2: Unselected Hallucinators
Group 3: Selected Non-Hallucinators
Group 4: Selected Hallucinators
Group 5: Controls

Table 5.2

Summary Statistics for Unselected Non-Hallucinators

Variable	Mean	SD	Median	Range	b_1	b_2
Colored Progressive Matrices	2.7	2.4	2.0	0-9	0.82*	-.05
Oral Word Fluency	12.1	3.7	12.3	5-18.7	-.12	-.98
Purdue Pegboard:						
Preferred	13.7	2.0	14.0	9-18	-.28	-.39
Non-Preferred	13.3	1.6	13.0	9-17	-.16	.02
Both	11.0	1.4	11.0	7-14	-.83*	.67
Assemblies	35.3	5.3	36.0	21-46	-.49	.52
L.J. Tactile Recognition:						
Preferred	41.0	40.7	29.1	8.1-277.3	4.35*	23.07*
Non-Preferred	31.8	19.2	27.9	9.1-81.7	1.23*	.94
Both	26.8	19.3	21.8	9.3-115.2	2.77*	9.33*
Wisconsin Card Sorting Test:						
Total Errors	24.7	19.2	19.0	2-90	1.13*	1.17
# of Categories	4.9	1.9	6.0	0-6	-1.52*	1.93
Speech Sounds Perception Test	5.2	3.4	4.0	0-16	1.31*	1.97
Trail Making Test:						
Part A	27.0	9.3	25.4	14.3-53.1	1.09*	.76
Part B	74.7	49.8	59.7	31.8-338.1	3.62*	16.32*
Halstead Category Test	53.9	30.2	51.5	7-128	.55*	-.26
Finger Tapping Test:						
Preferred	48.0	8.6	48.7	25.8-66.8	-.62*	.53
Non-Preferred	42.0	7.2	42.7	23.0-59.8	-.14	.27
Dynamometer:						
Preferred	41.5	14.5	44.8	13.0-63.3	-.24	-1.07
Non-Preferred	38.3	14.0	41.6	12.3-58.0	-.43	-1.13
Seashore Rhythm Test	4.3	3.6	3.5	0-15	1.43*	1.62
Finger Localization:						
Preferred Single Stimulation	.4	.93	0	0-4	2.92*	8.42*
Preferred Double Stimulation	2.5	2.9	2.0	0-16	2.36*	8.21*
Non-Preferred Single Stimulation	1.2	1.7	0	0-8	1.97*	4.72*
Non-Preferred Double Stimulation	3.6	3.7	3.0	0-17	1.71*	3.59

Table 5.2 (continued)

Variable	Mean	SD	Median	Range	b_1	b_2
WAIS-R:						
VIQ	98.7	14.5	98.5	53-129	-.52	.83
PIQ	96.6	13.8	95.5	68-131	.14	-.08
FSIQ	97.5	13.2	99.5	62-130	-.44	.69
Information	8.9	3.2	9.0	2-16	.26	-.01
Comprehension	9.8	3.3	10.0	2-18	-.19	-.05
Arithmetic	9.6	3.3	9.0	2-17	.00	-.56
Similarities	10.1	2.4	10.0	3-15	-.38	.57
Digit Span	9.0	2.9	9.0	3-16	.16	.04
Vocabulary	9.8	2.8	10.0	2-17	-.05	.33
Digit Symbol	7.8	2.3	8.0	4-13	.31	-.86
Picture Completion	9.3	2.4	10.0	4-17	.13	1.06
Block Design	10.0	2.8	10.0	5-16	.20	-.78
Picture Arrangement	9.2	3.2	8.5	4-17	.51	-.14
Object Assembly	9.8	2.8	10.0	4-18	.59*	.68

Note: $b_1$denotes Skewness, $b_2$denotes Kurtosis

*Denotes values of Skewness or Kurtosis in excess of the 95 percentile (Bowman & Shenton, 1975)

Table 5.3

Summary Statistics for Unselected Hallucinators

Variable	Mean	SD	Median	Range	b_1	b_2
Colored Progressive Matrices	3.5	2.4	3.0	0-8	.25	-.96
Oral Word Fluency	10.0	3.7	9.7	3.7-18.3	.34	-.22
Purdue Pegboard:						
Preferred	13.3	2.4	13.0	8-18	-.07	-.13
Non-Preferred	12.5	2.0	12.0	7-18	.02	.75
Both	10.1	1.7	10.0	6-15	-.18	1.07
Assemblies	30.4	6.6	30.0	16-48	.15	-.14
L.J. Tactile Recognition:						
Preferred	44.6	25.2	36.1	16.1-123.2	1.42*	1.55
Non-Preferred	46.1	33.6	39.2	11.0-192.5	2.28*	6.56*
Both	39.6	21.9	36.1	9.1-114.0	1.15*	1.90
Wisconsin Card Sorting Test:						
Total Errors	35.7	22.3	37.0	2-89	.27	-.55
# of Categories	3.9	2.2	5.0	0-6	-.42	-1.49
Speech Sounds Perception Test	6.6	4.3	5.0	1-22	1.32*	1.97
Trail Making Test:						
Part A	37.3	22.1	33.9	14.0-167.3	4.07*	22.43*
Part B	118.5	96.1	85.7	31.9-602.3	2.94*	11.63*
Halstead Category Test	74.7	33.9	76.0	7-133	-2.0*	-.67
Finger Tapping Test:						
Preferred	44.6	9.8	47.0	22.6-61.6	-.64*	-.12
Non-Preferred	40.0	8.2	41.6	19.2-54.2	-.54*	-.38
Dynamometer:						
Preferred	35.8	12.6	34.0	12.8-61.8	.17	-1.00
Non-Preferred	33.9	12.4	33.3	13.3-67.5	.30	-.40
Seashore Rhythm Test	5.8	4.2	5.0	0-15	.55*	-.78
Finger Localization:						
Preferred Single Stimulation	1.1	1.7	0.0	0-8	1.87*	4.12*
Preferred Double Stimulation	4.6	3.5	4.0	0-18	1.23*	2.54
Non-Preferred Single Stimulation	1.3	1.7	1.0	0-6	1.53*	1.61
Non-Preferred Double Stimulation	6.1	4.3	5.0	0-18	.93*	.39

Table 5.3 (continued)

Variable	Mean	SD	Median	Range	b_1	b_2
WAIS-R:						
VIQ	88.4	1387	91.0	53-113	-.33	-.48
PIQ	87.3	13.4	86.0	62-113	.13	-1.04
FSIQ	86.9	13.2	86.0	57-113	-.09	-.66
Information	7.7	3.1	7.0	2-14	-.07	-1.01
Comprehension	7.6	3.0	7.0	2-14	.25	-.58
Arithmetic	7.6	2.4	7.0	2-13	.19	-.71
Similarities	8.5	3.0	8.0	3-15	.18	-.63
Digit Span	7.8	2.4	8.0	2-13	.06	-.05
Vocabulary	8.4	2.8	9.0	2-17	.31	.51
Digit Symbol	6.7	2.4	6.0	3-12	.41	-.67
Picture Completion	8.1	2.8	8.0	2-14	.33	.02
Block Design	8.7	2.8	8.0	4-16	.49	-.05
Picture Arrangement	8.2	3.0	7.5	4-17	.66*	.23
Object Assembly	8.1	2.7	8.0	2-15	.17	-.25

Note: b_1 denotes Skewness, b_2 denotes Kurtosis

*Denotes values of Skewness or Kurtosis in excess of the 95 percentile (Bowman & Shenton, 1975)

Table 5.4

Summary Statistics for Selected Non-Hallucinators

Variable	Mean	SD	Median	Range	b_1	b_2
Colored Progressive Matrices	3.0	2.3	2.5	0-8	1.00*	.12
Oral Word Fluency	10.1	4.3	11.0	2.3-18	-.14	-.83
Purdue Pegboard:						
Preferred	13.8	2.7	14.0	7-19	-.24	-.11
Non-Preferred	13.2	2.0	13.0	9-18	.16	-.26
Both	11.0	2.2	11.0	6-17	.23	.61
Assemblies	27.9	6.4	28.0	17-43	.33	-.44
L.J. Tactile Recognition:						
Preferred	73.3	68.9	48.5	20.5-394.8	3.33*	14.28*
Non-Preferred	52.7	34.0	41.0	10.8-150.0	1.55*	2.09
Both	48.4	27.1	43.4	13.3-128.0	1.01*	.91
Wisconsin Card Sorting Test:						
Total Errors	37.7	22.9	41.0	2-82	.05	-1.15
# of Categories	4.1	2.3	6.0	0-6	-.58	-1.31
Speech Sounds Perception Test	8.0	4.6	7.0	2-19	.79*	-.31
Trail Making Test:						
Part A	39.3	17.4	33.9	18.7-108.4	2.08*	6.40*
Part B	128.7	114.6	102.4	36.1-687.8	3.82*	17.64*
Halstead Category Test	76.1	32.4	82.5	19-132	-.23	-.77
Finger Tapping Test:						
Preferred	43.1	8.4	44.8	27.4-55.8	-.36	-1.06
Non-Preferred	38.5	7.9	41.0	22.0-54.2	-.09	-.75
Dynamometer:						
Preferred	35.8	13.8	34.1	12.8-65.5	.48	-.62
Non-Preferred	32.3	13.6	28.8	9.0-63.0	.47	-.76
Seashore Rhythm Test	6.3	4.1	5.5	0-14	.38	-1.02
Finger Localization:						
Preferred Single Stimulation	1.4	2.1	0	0-10	2.47*	8.24*
Preferred Double Stimulation	4.6	4.8	3.5	0-25	2.53*	9.30*
Non-Preferred Single Stimulation	1.6	2.4	1.0	0-12	2.62*	9.16*
Non-Preferred Double Stimulation	6.6	4.4	7.0	0-20	.78*	1.24

Table 5.4 (continued)

Variable	Mean	SD	Median	Range	b_1	b_2
WAIS-R:						
VIQ	92.6	16.0	93.0	59-122	-.36	-.28
PIQ	84.7	14.6	84.5	51-123	.18	.79
FSIQ	88.4	14.8	88.5	53-124	.01	.43
Information	9.4	2.8	10.0	3-15	-.43	-.25
Comprehension	8.2	3.6	8.0	1-15	-.05	-.38
Arithmetic	8.8	3.4	8.5	4-16	.34	-1.06
Similarities	8.6	3.9	10.0	1-14	-.41	-.82
Digit Span	8.5	2.6	8.0	4-17	1.05*	2.95
Vocabulary	8.4	3.4	8.5	1-15	-.33	-.28
Digit Symbol	6.1	2.0	5.5	3-12	1.25*	1.78
Picture Completion	6.1	3.0	6.0	1-13	.19	-.23
Block Design	9.0	3.2	9.0	1-16	-.13	.24
Picture Arrangement	7.3	3.7	7.0	1-16	.67	-.15
Object Assembly	8.7	3.5	9.0	1-16	-.31	.41

Note: $b_1$denotes Skewness, $b_2$denotes Kurtosis

*Denotes values of Skewness or Kurtosis in excess of the 95 percentile (Bowman & Shenton, 1975)

Table 5.5

Summary Statistics for Selected Hallucinators

Variable	Mean	SD	Median	Range	b_1	b_2
Colored Progressive Matrices	4.4	3.0	4.0	1-10	.52	-.97
Oral Word Fluency	9.4	4.7	8.5	2.3-19.0	.41	-.61
Purdue Pegboard:						
Preferred	13.6	3.2	14.5	8-20	-.14	-.54
Non-Preferred	12.9	2.3	13.5	9-17	-.33	-.60
Both	11.2	2.2	11.5	7-15	-.21	-.89
Assemblies	26.9	8.4	27.0	12-41	-.15	-.72
L.J. Tactile Recognition:						
Preferred	51.3	21.8	52.9	16.3-93.8	.02	-.84
Non-Preferred	47.2	25.5	41.1	11.4-91.9	.36	-1.25
Both	44.0	34.4	31.8	9.7-150.2	1.81*	3.75
Wisconsin Card Sorting Test:						
Total Errors	34.2	21.4	32.5	3-91	.70	1.12
# of Categories	3.9	2.7	6.0	0-6	-.52	-1.81
Speech Sounds Perception Test	10.0	6.1	9.0	2-26	1.35*	2.03
Trail Making Test:						
Part A	74.0	92.0	41.1	20.4-360.8	2.6*	6.12*
Part B	164.2	186.6	86.8	42.7-800.7	2.47*	6.71*
Halstead Category Test	82.7	38.3	84.5	15-133	-.31	-1.14
Finger Tapping Test:						
Preferred	39.5	10.9	37.9	23.2-61.2	.39	-.64
Non-Preferred	35.7	9.8	36.2	22.6-59.2	.74	.61
Dynamometer:						
Preferred	34.7	13.3	33.5	12.0-55.3	.13	-1.06
Non-Preferred	31.9	14.3	31.1	10.0-54.1	.13	-1.21
Seashore Rhythm Test	6.7	4.5	6.0	2-14	.47	-1.25
Finger Localization:						
Preferred Single Stimulation	1.4	2.7	.5	0-11	3.03*	9.68*
Preferred Double Stimulation	5.5	5.2	4.0	0-22	1.75*	4.33*
Non-Preferred Single Stimulation	1.6	3.2	1.0	0-14	3.52*	13.83*
Non-Preferred Double Stimulation	5.6	6.9	3.5	0-31	2.86*	10.06*

Table 5.5 (continued)

Variable	Mean	SD	Median	Range	b_1	b_2
WAIS-R:						
VIQ	88.0	13.3	87.5	60-108	-.22	-.64
PIQ	82.5	10.9	82.0	63-108	.15	.72
FSIQ	85.2	11.1	84.0	59-102	-.41	.02
Information	8.8	2.8	9.0	3-14	-.20	-.04
Comprehension	7.0	2.6	6.5	1-12	.00	.59
Arithmetic	7.8	3.4	8.0	2-16	.37	.54
Similarities	8.1	4.0	8.0	2-17	.39	.30
Digit Span	7.8	2.4	7.5	1-14	-.24	4.68*
Vocabulary	7.6	2.0	8.0	3-12	-.40	1.52
Digit Symbol	6.4	2.2	5.5	4-13	1.78*	3.58
Picture Completion	5.4	2.8	5.0	1-10	.14	-1.15
Block Design	7.4	2.9	7.5	3-12	-.04	-1.16
Picture Arrangement	5.9	3.0	5.0	1-11	.15	-1.21
Object Assembly	8.6	2.7	8.0	4-13	.01	-.93

Note: $b_1$denotes Skewness, $b_2$denotes Kurtosis

*Denotes values of Skewness or Kurtosis in excess of the 95 percentile (Bowman & Shenton, 1975)

Table 5.6

Summary Statistics for Controls

Variable	Mean	SD	Median	Range	b_1	b_2
Colored Progressive Matrices	1.2	1.3	1.0	0-6	1.17*	1.16
Oral Word Fluency	14.6	4.0	14.0	7.3-29.3	.86*	.90
Purdue Pegboard:						
Preferred	16.3	1.9	16.0	11-22	.22	.46
Non-Preferred	15.5	1.8	16.0	12-20	-.27	-.22
Both	13.1	1.5	13.0	9-17	-.11	-.16
Assemblies	40.2	5.2	40.0	22-56	-.15	.72
L.J. Tactile Recognition:						
Preferred	33.9	20.8	29.0	6.5-138.0	1.90*	5.41*
Non-Preferred	31.9	25.5	24.0	7.0-211.0	3.40*	16.82*
Both	22.2	14.0	18.7	4.3-109.0	2.17*	5.95*
Wisconsin Card Sorting Test:						
Total Errors	13.5	14.3	8.0	0-78	2.15*	4.51*
# of Categories	5.8	1.0	6.0	0-6	-4.19*	17.00*
Speech Sounds Perception Test	4.1	2.3	4.0	0-15	1.20*	3.11
Trail Making Test:						
Part A	24.2	7.8	22.6	11.8-52.7	1.09*	1.51
Part B	51.2	15.6	48.1	21.0-111.0	.85*	1.02
Halstead Category Test	35.4	17.9	33.5	6-106	.75*	.72
Finger Tapping Test:						
Preferred	49.1	8.1	49.0	26.6-70.0	.08	.03
Non-Preferred	44.8	6.9	45.2	26.8-63.4	.12	.08
Dynamometer:						
Preferred	41.8	13.0	39.0	17.8-83.0	.56*	-.32
Non-Preferred	39.0	12.8	35.9	16.0-73.0	.50*	-.62
Seashore Rhythm Test	2.5	2.0	2.0	0-9	1.08*	1.12
Finger Localization:						
Preferred Single Stimulation	.40	.80	0	0-4	1.92*	3.50
Preferred Double Stimulation	2.0	2.0	2.0	0-12	1.50*	3.56
Non-Preferred Single Stimulation	.5	.80	0	0-4	1.86*	2.90
Non-Preferred Double Stimulation	2.4	2.4	2.0	0-16	2.01*	6.73*

Table 5.6 (continued)

Variable	Mean	SD	Median	Range	b_1	b_2
WAIS-R:						
VIQ	112.7	10.4	113.0	81-147	.06	.41
PIQ	108.9	10.3	109.0	85-139	.06	-.11
FSIQ	111.9	9.5	112.0	87-142	.15	.16
Information	11.4	2.3	12.0	4-16	-.61*	.24
Comprehension	11.6	2.6	11.0	5-18	-.05	-.69
Arithmetic	11.8	2.5	12.0	6-17	-.04	-.48
Similarities	11.9	3.0	11.0	3-19	-.06	.09
Digit Span	11.5	2.8	11.0	5-18	.39*	-.10
Vocabulary	11.6	2.7	11.0	6-18	.47*	-.46
Digit Symbol	11.1	2.7	11.0	5-19	.45*	-.11
Picture Completion	10.0	2.8	10.0	2-18	.13	.56
Block Design	12.0	2.4	12.0	6-17	.10	-.66
Picture Arrangement	11.4	2.7	11.0	4-18	.12	-.38
Object Assembly	11.1	2.7	11.0	1-17	-.04	.46

Note: b_1 denotes Skewness, b_2 denotes Kurtosis

*Denotes values of Skewness or Kurtosis in excess of the 95 percentile (Bowman & Shenton, 1975)

Table 5.7

Kolmogorov-Smirnov Goodness of Fit for Normality, Two-Tailed P Values

Variable	Group 1	Group 2	Group 3	Group 4	Group 5
Colored Progressive Matrices	.19	.13	.11	.52	.00*
Oral Word Fluency	.75	.96	.51	.96	.10
Purdue Pegboard:					
Preferred	.31	.31	.95	.58	.00*
Non-Preferred	.11	.42	.53	.47	.00*
Both	.07	.03*	.81	.78	.00*
Assemblies	.74	.91*	.78	.98	.13
L.T. Tactile Recognition:					
Preferred	.01*	.05*	.07	.77	.00*
Non-Preferred	.26*	.05*	.13	.45	.00*
Both	.01*	.67*	.64	.58	.00*
Wisconsin card Sorting Test:					
Total Errors	.23*	.49*	.81	.95	.00*
# of Categories	.00*	.01*	.00*	.01*	.00*
Speech Sounds Perception Test	.04*	.04	.24	.50	.00*
Trail Making Test:					
Part A	.31	.13*	.21	.03*	.06*
Part B	.03*	.04*	.04*	.05*	.15
Halstead Category Test	.69	.98	.81	.97	.16*
Finger Tapping Test:					
Preferred	.62	.23	.72	.97	.80
Non-Preferred	.90	.45	.48	.85	.91
Dynamometer:					
Preferred	.60	.33	.93	.93	.08
Non-Preferred	.12	.82	.32	.94	.01*
Seashore Rhythm Test	.02*	.14	.44	.58	.00*
Finger Localization:					
Preferred Single Stimulation	.00*	.00*	.01*	.01*	.00*
Preferred Double Stimulation	.00*	.27	.22	.32	.00*
Non-Preferred Single Stimulation	.00*	.00*	.01*	.03*	.00*
Non-Preferred Double Stimulation	.13	.10	.96	.35	.00*

Table 5.7 (continued)

Variable	Group 1	Group 2	Group 3	Group 4	Group 5
WAIS-R:					
VIQ	.96	.82	.99	.78	.67
PIQ	.99	.45	.96	.99	.94
FSIQ	.73	.66	.81	.92	.75
Information	.27	.09	.14	.86	.00*
Comprehension	.87	.63	.64	.70	.00*
Arithmetic	.77	.10	.43	.98	.16
Similarities	.66	.39	.14	.90	.00*
Digit Span	.45	.08	.27	.26	.00*
Vocabulary	.65	.54	.62	.49	.00*
Digit Symbol	.20	.08	.07	.19	.02*
Picture Completion	.31	.52	.33	.71	.00*
Block Design	.40	.39	.74	.89	.00*
Picture Arrangement	.20	.11	.25	.53	.01*
Object Assembly	.05*	.41	.27	.95	.04*

*p.05

thus prior to further analysis data were transformed to ranks. Following this, parametric analyses were applied to the ranks of the data. This is the procedure recommended by Conover (1980) and Conover and Iman (1981) who have found it to be robust with respect to the analysis of experimental data that is not normally distributed.

Analysis of variance of WAIS-R Verbal IQ by group membership, sex, and age band showed significant main effects for group membership (p<.000), sex (p<.028), and age band (p<.000) (see Table 5.8). A similar analysis for Performance IQ showed group membership (p<.000) and sex (p<.030) to be significant, but not age band (see Table 5.9). For Full Scale IQ, group membership (p<.000), sex (p<.020), and age band (p<.007) were all significant (see Table 15.10). There were no two-way or three-way interactions for Verbal IQ, Performance IQ, or Full Scale IQ, with the exception of a group by age band interaction for Performance IQ (p<.028). The effect for sex was less significant than the effects for age and group membership. Results are due to the higher IQ's of controls and the fact that controls were, on average, younger than the patients.

5.4 Multivariate Analysis of Variance

Multivariate analysis of variance was performed on the rank transformed data with estimated missing values.

Table 5.8

WAIS-R Verbal IQ by Group Membership, Sex, and Age Band

Source of Variation	Sum of Squares	DF	Mean Square	F	P
Main Effects	40811.9	6	6802.0	45.5	.000
Group	40147.4	4	10036.9	67.2	.000
Sex	732.6	1	732.6	4.9	.028
Age Band	2050.2	1	2050.2	13.7	.000
Two Way Interactions	1190.5	9	132.7	.9	.539
Group by Sex	325.8	4	81.4	.5	.703
Group by Age Band	867.8	4	217.0	1.5	.217
Sex by Age Band	82.8	1	82.8	.6	.457
Three-Way Interactions	250.2	4	62.6	.4	.795
Group by Sex by Age Band	250.2	4	62.6	.4	.795
Explained	42252.6	19	2223.8	14.9	.000
Residual	48276.3	323	149.5		
Total	90528.9	342	264.7		

Table 5.9

WAIS-R Performance IQ by Group Membership, Sex, and Age Band

Source of Variation	Sum of Squares	DF	Mean Square	F	P
Main Effects	37760.7	6	6293.5	45.3	.000
Group	35363.1	4	8840.8	63.7	.000
Sex	613.0	1	613.0	4.4	.036
Age Band	31.0	1	31.0	.2	.637
Two Way Interactions	1924.2	9	213.8	1.5	.133
Group by Sex	589.1	4	147.3	1.1	.376
Group by Age Band	1530.3	4	382.6	2.8	.028
Sex by Age Band	25.7	1	25.7	.2	.667
Three-Way Interactions	320.2	4	80.0	.6	.680
Group by Sex by Age Band	320.2	4	80.06	.6	.680
Explained	40005.1	19	2105.5	15.2	.000
Residual	44300.6	319	138.9		
Total	84305.7	338	249.4		

Table 5.10

WAIS-R Full Scale IQ by Group Membership, Sex, and Age Band

Source of Variation	Sum of Squares	DF	Mean Square	F	P
Main Effects	44087.6	6	7347.9	57.7	.000
Group	43032.6	4	10758.1	84.5	.000
Sex	695.3	1	695.3	5.5	.020
Age Band	945.3	1	945.3	7.4	.007
Two Way Interactions	1133.7	9	126.0	1.0	.448
Group by Sex	207.9	4	52.0	.4	.803
Group by Age Band	994.0	4	248.5	3.0	.102
Sex by Age Band	58.4	1	58.4	.5	.499
Three-Way Interactions	276.5	4	69.1	.5	.704
Group by Sex by Age Band	276.5	4	69.1	.5	.704
Explained	45497.8	19	2394.6	18.8	.000
Residual	40603.1	319	127.3		
Total	86100.9	334	254.7		

Results for main effects and interactions by group membership, sex, and age band are reported in Table 5.11. It can be seen that age, sex, and group membership are all highly significant, but that there are no two-way or three-way interactions between these variables. In order to determine which neuropsychological variables contributed to these main effects, Scheffe contrasts were used. Data are reported in Table 5.12 which presents Scheffe contrasts by group membership, then in Tables 5.13 to 5.16 which give Scheffe contrasts for groups of young females, old females, young males, and old males respectively. Results in Table 5.12 show a significant main effect for all the neuropsychological and Wechsler variables. In all cases, patients differ from controls, in that they are ranked more highly and hence show greater impairment of functioning.

5.4.1 Scheffe Contrasts, All Groups

Inspection of Table 5.12 shows that for nearly every variable the patient groups differ from controls. Exclusive of patient/control differences it can be seen that Group 1, the unselected non-hallucinators, differs from the other three patient groups on several variables. Group 1, the unselected non-hallucinators, differs from Group 2, the unselected hallucinators, on Purdue Pegboard - Assemblies, L.J. Tactile Recognition - Both Hand Trial, Wisconsin Card Sorting Test - number of categories sorted, Trail Making Test - Part A and B, Halstead Category Test -

number of errors, Finger Localization Preferred Hand - Double Stimulation Errors, and on the Comprehension and Arithmetic subtests of the WAIS-R. This group also differs from Group 3, the selected non-hallucinators on Purdue Pegboard - Assemblies, L.J. Tactile Recognition - Preferred, Nonpreferred and Both Hand Trials, Trail Making Test - Part A and B, Halstead Category Test- number of errors, Finger Localization Nonpreferred Hand - Double Stimulation Errors, and on the Digit Symbol and Picture Completion Symbol subtests of the WAIS-R. In addition, the unselected non-hallucinators differ from Group 4, the selected hallucinators, on the Purdue Pegboard-Assemblies, Trail Making Test - Part A, Finger Tapping - Preferred Hand, Speech Sounds Perception Test, and the Comprehension, Vocabulary, Picture Completion, Block Design, and Picture Arrangement subtests of the WAIS-R. Lastly, it is noted that Group 2, the unselected hallucinators, differs from Group 4, the selected hallucinators, on the Picture Completion subtest of the WAIS-R and that Group 3, the selected non-hallucinators, differs from Group 4, the selected hallucinators, on the Wisconsin Card Sorting Test - number of errors. To summarize these differences, it is evident that Group 1 is less severely impaired (i.e., has a lower rank order group mean) that the other patient groups. These results are based on a mixture of motor, cognitive, and sensory-perceptual tests, which do not define any clear

Table 5.11

Multivariate Analysis of Variance of Main Effects and Interactions by Group Membership, Sex, and Age Band

Effect	Test Name	Value	F	Hypoth DF	Error DF	Significance of F
Group	Wilks Lambda	.23	3.68	140.0	1153.4	.000
Sex	Wilks Lambda	.35	15.58	35.0	289.0	.000
Age Band	Wilks Lambda	.78	2.37	35.0	289.0	.000
Group by Sex	Wilks Lambda	.66	.92	140.0	1153.4	.731
Group by Age Band	Wilks Lambda	.62	1.07	140.0	1153.4	.296
Sex by Age Band	Wilks Lambda	.87	1.28	35.0	289.0	.144
Group by Sex by Age Band	Wilks Lambda	.63	1.03	140.0	1153.4	.408

Table 5.12

Scheffe Contrasts by Group Membership for all Neuropsychological Variables

Variable	Significance of Main Effect	Rank Order Group Mean 1	2	3	4	5	Significant Group Differences[a]
CPM	.000	191.9	232.1	215.8	255.0	130.0	1-5, 2-5, 3-5, 4-5
OWF	.000	185.7	236.6	225.0	241.0	131.1	1-5, 2-5, 3-5, 4-5
PURDP	.000	226.7	239.6	215.1	217.6	122.9	1-5, 2-5, 3-5, 4-5
PURDNP	.000	220.5	248.7	221.5	228.2	119.5	1-5, 2-5, 3-5, 4-5
PURDB	.000	222.3	262.2	220.1	206.7	117.6	1-5, 2-5, 3-5, 4-5
PURDA	.000	188.9	246.1	272.4	270.5	115.2	1-5, 2-5, 3-5, 4-5, 1-2, 1-3, 1-4
LJPTA	.000	162.1	196.3	242.5	230.1	147.9	2-5, 3-5, 4-5, 1-3
LJNPT	.000	157.4	208.3	233.6	218.3	148.6	2-5, 3-5, 4-5, 1-3
LJBT	.000	163.8	230.3	254.5	222.6	135.8	2-5, 3-5, 4-5, 1-2, 1-3
WCST	.000	193.6	233.2	241.7	231.1	127.9	1-5, 2-5, 3-4, 4-5
WCSCAT	.000	182.9	228.2	209.3	204.0	141.4	1-5, 2-5, 3-5, 4-5, 1-2
SPEECHS	.000	174.2	202.3	233.9	264.2	140.5	2-5, 3-5, 4-5, 1-4
TRAILA	.000	161.6	226.9	248.0	260.0	134.5	2-5, 3-5, 4-5, 1-2, 1-3, 1-4
TRAILB	.000	185.6	244.6	263.3	243.8	121.3	1-5, 2-5, 3-5, 4-5, 1-2, 1-3
HALCAT	.000	183.6	240.6	245.2	251.8	125.5	1-5, 2-5, 3-5, 4-5, 1-2, 1-3
FTAPPT	.000	159.6	193.9	214.0	242.9	153.3	3-5, 4-5, 1-4
FTAPNP	.000	179.5	196.7	218.6	262.5	144.8	2-5, 3-5, 4-5
DYNP							N.S.
DYNNP							N.S.

Table 5.12 (continued)

Variable	Significance of Main Effect	Rank Order Group Mean 1	2	3	4	5	Significant Group Differences[a]
SEAR	.000	186.8	214.2	235.9	238.3	134.3	1-5, 2-5, 3-5, 4-5
FLOCPSS							N.S.
FLOCPDS	.000	156.7	227.7	214.3	229.3	145.3	2-5, 3-5, 4-5, 1-2
FLOCNPSS	.000	190.8	195.9	206.2	198.7	150.1	2-5, 3-5
FLOCNPDS	.000	168.7	234.7	245.0	198.9	137.4	2-5, 3-5, 1-2, 1-3
INFO	.000	214.7	242.6	195.7	218.9	128.9	1-5, 2-5, 3-5, 4-5
COMP	.000	180.5	245.3	223.8	203.8	127.7	1-5, 2-5, 3-5, 4-5, 1-2, 1-4
ARIT	.000	191.5	252.8	214.2	243.4	126.3	1-5, 2-5, 3-5, 4-5, 1-2
SIMI	.000	186.2	234.7	216.5	240.3	133.2	1-5, 2-5, 3-5, 4-5
DSPA	.000	200.8	245.0	223.9	250.0	123.5	1-5, 2-5, 3-5, 4-5
VOCA	.000	186.9	231.3	221.5	266.8	130.2	1-5, 2-5, 3-5, 4-5, 1-4
DSYM	.000	212.7	246.2	267.3	260.5	110.4	1-5, 2-5, 3-5, 4-5, 1-3
PCOM	.000	158.5	203.0	256.7	274.1	139.4	2-5, 3-5, 4-5, 1-3, 1-4, 2-4
BDES	.000	192.9	236.3	221.3	265.1	127.2	1-5, 2-5, 3-5, 4-5, 1-4
PARR	.000	193.2	222.4	241.3	275.2	126.4	1-5, 2-5, 3-5, 4-5, 1-4
OASS	.000	183.1	234.9	212.3	220.9	136.9	1-5, 2-5, 3-5, 4-5

NOTE. Group 1 = Unselected Non-Hallucinators
Group 2 = Unselected Hallucinators
Group 3 = Selected Non-Hallucinators
Group 4 = Selected Hallucinators
Group 5 = Controls

[a] Scheffe contrasts set at $p < .05$

Table 5.13

Scheffe Contrasts for Young Females (15-30 years) for all Neuropsychological Variables

Variable	Rank Order Group Mean 1	2	3	4	5	Significant Group Differences[a]
CPM	171.5	242.9	202.3	160.0	128.1	2-5
OWF	182.0	247.2	255.6	197.0	143.2	2,5, 3-5
PURDP	213.0	252.5	225.5	110.9	94.2	1-5, 2-5, 3-5
PURDNP	200.1	265.8	185.5	149.5	98.6	1-5, 2-5
PURDB	206.6	247.6	221.6	138.5	88.7	1-5, 2-5, 3-5
PURDA	141.0	238.4	264.2	211.1	87.4	2-5, 3-5, 4-5, 1-3
LJPTA	92.9	229.8	229.5	195.5	159.8	1-2, 1-3
LJNPT						N.S.
LJBT	112.1	270.1	261.1	207.5	144.0	2-5, 3-5, 1-2, 1-3
WCST	133.7	215.3	259.9	191.0	120.2	2-5, 3-5, 1-3
WCSCAT	124.5	229.9	242.0	177.6	139.2	2-5, 3-5, 1-2, 1-3
SPEECHS						N.S.
TRAILA	118.7	238.4	290.2	273.9	141.7	3-5, 1-3
TRAILB	139.8	261.6	283.2	191.3	116.4	2-5, 3-5, 1-2, 1-3
HALCAT	160.8	243.9	245.1	191.4	132.2	2-5, 3-5
FTAPPT						N.S.
FTAPNP	258.2	288.4	248.6	287.4	182.7	2-5
DYNP	277.2	287.4	302.3	301.3	237.8	3-5
DYNNP	265.0	269.7	305.5	313.3	236.5	3-5, 4-5

Table 5.13 (continued)

Variable	Rank Order Group Mean 1	2	3	4	5	Significant Group Differences[a]
SEAR						N.S.
FLOCPSS	168.2	244.5	140.8	114.0	154.1	2-5
FLOCPDS	161.0	267.2	213.9	158.3	138.2	2-5
FLOCNPSS	203.0	251.3	150.4	138.8	148.7	2-5
FLOCNPDS	108.8	263.9	285.1	161.1	116.8	2-5, 3-5
INFO	266.8	270.4	194.3	255.3	172.0	1-5, 1-2
COMP	206.1	259.7	275.4	282.8	143.6	2-5, 3-5, 4-5
ARIT	200.0	296.8	276.9	245.5	162.4	2-5, 3-5
SIMI	204.1	254.9	186.9	264.8	129.5	2-5
DSPA	174.3	251.6	236.7	219.5	139.2	2-5, 3-5
VOCA	210.3	248.4	225.2	261.6	155.5	2-5
DSYM	157.3	232.7	282.5	204.1	86.8	1-5, 2-5, 3-5, 4-5, 1-3
PCOM	171.8	203.4	274.1	262.9	170.9	3-5
BDES	174.4	252.9	211.0	272.3	133.4	2-5, 4-5
PARR	219.2	248.8	233.9	251.5	133.1	1-5, 2-5, 3-5
OASS	154.2	265.2	207.9	213.6	144.5	2-5

NOTE. Group 1 = Unselected Non-Hallucinators
Group 2 = Unselected Hallucinators
Group 3 = Selected Non-Hallucinators
Group 4 = Selected Hallucinators
Group 5 = Controls

[a] Scheffe contrasts set at $p < .05$

Table 5.14

Scheffe Contrasts for Older Females (31-45 years) for all Neuropsychological Variables

Variable	Rank Order Group Mean 1	2	3	4	5	Significant Group Differences[a]
CPM	203.8	264.3	191.4	286.7	120.2	2-5, 4-5
OWF						N.S.
PURDP						N.S.
PURDNP	228.9	268.2	247.4	280.1	121.0	2-5, 3-5, 4-5
PURDB	232.3	267.5	222.8	225.5	116.5	2-5
PURDA	186.8	253.2	283.3	288.4	131.1	2-5, 3-5, 4-5
LJPTA	211.6	221.8	287.6	255.0	151.5	3-5
LJNPT						N.S.
LJBT	194.3	272.7	298.0	222.8	170.2	2-5, 3-5, 1-3
WCST						N.S.
WCSCAT						N.S.
SPEECHS						N.S.
TRAILA	223.9	268.8	259.4	257.7	156.3	2-5
TRAILB	230.6	274.9	266.9	255.8	166.3	2-5
HALCAT	248.8	285.0	251.7	261.8	161.3	2-5
FTAPP						N.S.
FTAPNP						N.S.
DYNP	275.3	300.4	277.2	279.3	229.2	2-5
DYNNP	287.5	298.6	295.2	281.5	234.5	2-5, 3-5

Table 5.14 (continued)

Variable	Rank Order Group Mean 1	2	3	4	5	Significant Group Differences[a]
SEAR	175.4	244.3	276.3	269.5	122.3	2-5, 3-5, 4-5
FLOCPSS	114.0	139.5	241.2	217.0	126.0	5-3, 1-3, 2-3
FLOCPDS						N.S.
FLOCNPSS						N.S.
FLOCNPDS						N.S.
INFO						N.S.
COMP	159.4	234.5	194.2	262.0	81.6	2-5, 4-5
ARIT	172.5	250.2	201.7	259.4	127.0	2-5
SIMI						N.S.
DSPA	185.0	227.4	237.0	270.0	109.6	2-5, 3-5, 4-5
VOCA	135.8	196.6	202.7	282.8	96.0	4-5
DSYM	217.2	238.0	260.4	279.4	92.4	1-5, 2-5, 3-5, 4-5
PCOM						N.S.
BDES	243.9	273.7	284.2	295.5	158.1	2-5, 3-5
PARR	232.7	241.0	284.7	296.2	117.9	2-5, 3-5, 4-5
OASS	237.2	262.9	277.3	257.4	136.1	2-5, 3-5

NOTE. Group 1 = Unselected Non-Hallucinators
Group 2 = Unselected Hallucinators
Group 3 = Selected Non-Hallucinators
Group 4 = Selected Hallucinators
Group 5 = Controls

[a] Scheffe contrasts set at $p < .05$

Table 5.15

Scheffe Contrasts for Young Males (15-30 years) for all Neuropsychological Variables

Variable	Rank Order Group Mean					Significant Group Differences[a]
	1	2	3	4	5	
CPM	192.6	215.9	240.7	297.8	134.3	2-5, 3-5, 4-5
OWF	203.4	252.4	233.2	278.8	132.4	2-5, 3-5, 4-5
PURDP	236.5	242.8	223.4	245.5	139.7	1-5, 2-5, 3-5
PURDNP	231.9	236.7	247.8	226.8	130.6	1-5, 2-5, 3-5
PURDB	225.4	259.9	235.0	242.7	130.2	1-5, 2-5, 3-5, 4-5
PURDA	198.5	240.2	285.4	294.9	120.5	1-5, 2-5, 3-5, 4-5
LJPTA						N.S.
LJNPT						N.S.
LJBT	174.5	196.6	225.7	229.3	129.9	3-5
WCST	192.6	235.8	260.3	281.2	134.3	2-5, 3-5, 4-5
WCSCAT	175.2	211.1	239.3	248.3	144.3	2-5, 3-5
SPEECHS	182.8	189.7	258.7	257.0	136.9	3-5
TRAILA	135.8	199.2	234.6	299.8	129.6	2-5, 3-5, 4-5, 1-4
TRAILB	185.4	224.2	270.5	287.9	111.5	1-5, 2-5, 3-5, 4-5
HALCAT	154.4	205.9	262.4	270.3	112.6	2-5, 3-5, 4-5, 1-3
FTAPPT						N.S.
FTAPNP	110.9	154.8	178.4	252.2	129.1	1-4
DYNP						N.S.
DYNNP						N.S.

Table 5.15 (continued)

Variable	Rank Order Group Mean 1	2	3	4	5	Significant Group Differences[a]
SEAR	185.6	234.7	237.0	265.9	137.5	2-5, 3-5, 4-5
FLOCPSS						N.S.
FLOCPDS	139.1	216.3	238.9	248.8	148.4	3-5
FLOCNPSS	190.3	200.3	247.0	251.3	155.7	3-5
FLOCNPDS	183.4	240.4	262.1	250.2	145.9	2-5, 3-5
INFO	219.3	244.1	211.0	242.2	101.8	1-5, 2-5, 3-5, 4-5
COMP	187.3	258.2	227.3	241.1	131.7	2-5, 3-5
ARIT	221.3	255.4	227.2	277.4	106.7	1-5, 2-5, 3-5, 4-5
SIMI	186.0	248.6	238.3	191.2	141.9	2-5, 3-5
DSPA	225.0	261.7	234.7	262.6	121.4	1-5, 2-5, 3-5, 4-5
VOCA	222.2	246.3	260.0	272.0	127.0	1-5, 2-5, 3-5, 4-5
DSYM	212.3	259.5	274.9	268.8	120.7	1-5, 2-5, 3,5, 4-5
PCOM	122.7	197.1	249.1	262.2	116.6	2-5, 3-5, 4-5, 1-3, 1-4
BDES	160.3	222.7	196.8	228.0	120.9	2-5
PARR	133.6	203.4	247.5	260.9	121.3	2-5, 3-5, 4-5, 1-3
OASS	157.3	228.4	182.5	208.4	133.0	2-5

NOTE. Group 1 = Unselected Non-Hallucinators
Group 2 = Unselected Hallucinators
Group 3 = Selected Non-Hallucinators
Group 4 = Selected Hallucinators
Group 5 = Controls

[a] Scheffe contrasts set at $p < .05$

Table 5.16

Scheffe Contrasts for Older Males (31-45 years) for all Neuropsychological Variables

Variable	Rank Order Group Mean					Significant Group Differences[a]
	1	2	3	4	5	
CPM						N.S.
OWF	196.5	228.1	206.2	224.6	104.4	1-5, 2-5
PURDP						N.S.
PURDNP	220.1	235.5	157.1	230.4	139.6	1-5
PURDB	225.9	277.3	174.1	202.8	156.4	2-5
PURDA	216.3	261.8	230.3	272.1	156.1	2-5, 4-5
LJPTA						N.S.
LJNPT						N.S.
LJBT						N.S.
WCST	232.5	265.0	190.7	234.0	119.5	1-5, 2-5
WCSCAT	228.9	260.9	124.5	202.1	126.1	1-5, 2-5, 2-3
SPEECHS	183.6	196.5	187.4	298.1	112.3	4-5
TRAILA						N.S.
TRAILB						N.S.
HALCAT	197.2	273.0	189.0	269.7	133.3	2-5, 4-5
FTAPP						N.S.
FTAPNP	180.2	124.3	176.5	204.7	74.5	1-5
DYNP	97.0	126.8	171.4	137.6	61.4	3-5
DYNNP	94.5	140.1	176.0	124.4	55.3	2-5, 3-5

Table 5.16 (continued)

Variable	Rank Order Group Mean 1	2	3	4	5	Significant Group Differences[a]
SEAR						N.S.
FLOCPSS						N.S.
FLOCPDS	175.6	192.9	166.8	280.1	133.9	4-5
FLOCNPSS						N.S.
FLOCNPDS						N.S.
INFO						N.S.
COMP	164.9	206.8	195.3	273.6	91.8	2-5, 4-5
ARIT						N.S.
SIMI	169.8	183.3	199.4	2525.4	100.5	4-5
DSPA	204.3	213.8	154.0	237.7	92.9	1-5
VOCA	159.6	221.2	149.9	246.3	86.8	2-5, 4-5
DSYM	252.5	232.8	238.8	274.8	151.3	1-5, 4-5
PCOM	170.3	193.8	194.8	281.9	115.4	4-5
BDES	213.8	199.4	186.2	259.9	115.1	1-5, 4-5
PARR	213.5	221.7	157.4	283.2	130.0	1-5, 4-5
OASS						N.S.

NOTE. Group 1 = Unselected Non-Hallucinators
Group 2 = Unselected Hallucinators
Group 3 = Selected Non-Hallucinators
Group 4 = Selected Hallucinators
Group 5 = Controls

[a] Scheffe contrasts set at $p < .05$

pattern of neuropsychological deficits.

5.4.2 Scheffe Contrasts, Young Females

Scheffe contrasts for young females aged 15 to 30 years are reported in table 5.13 and show that the main differences are between patients and controls. Within the patient subgroups, differences are present on several neuropsychological variables. Group 1 differs from Group 2 on L.J. Tactile Recognition - Preferred and Both Hands, Wisconsin Card Sorting Test - number of categories sorted, Trail Making Test - Part B, and the Information subtest of the WAIS-R. Group 1 also differs from Group 3 on Purdue Pegboard - Assemblies, L.J. Tactile Recognition - Preferred and Both Hand trials, Wisconsin Card Sorting - total errors and number of categories sorted, Tails A and B, and the Digit Symbol subtest of the WAIS-R.

5.4.3 Scheffe Contrasts, Older Females

Inspection of Scheffe contrasts for older females aged 31 to 45 years shows essentially patient/control differences (see Table 5.14). Within patient subgroup differences are present between Groups 1 and 3 on L.J. Tactile Recognition - Both Hand Trial and Finger Localization - Preferred Hand-Single Stimulation. The Finger Localization - Preferred Hand - Single Stimulation also discriminates between Group 2 and Group 3.

5.4.4 Scheffe Contrasts, Young Males

Table 5.15 presents Scheffe contrasts for young males aged 15 to 30 years. As with previous groupings patient/control differences predominate. In addition, Group 1 differs from Group 3 on the Halstead Category Test - number of errors, and the Picture Completion and Picture Arrangement subtests of the WAIS-R. Group 1 also differs from Group 4, on Trail Making - Part A, Finger Tapping - Nonpreferred Hand, and the Picture Completion subtest of the WAIS-R.

5.4.5 Scheffe Contrasts, Older Males

Examination of the Scheffe contrasts for older males aged 31 to 45 shows patient/control group differences, with the exception of the Wisconsin Card Sorting Test - number of categories sorted, which discriminates between Group 2 and Group 3 (see Table 5.16).

In summary, the results of the MANOVA and Scheffe contrasts indicate very clear patient/control differences. Within the patient groups much less discrimination was evident. Examination of the rank order group means in Table 5.12 shows that Group 1, the unselected non-hallucinators, differed from the other three patient groups in that they showed less severity of dysfunction. The rank scores were determined such that an increased score indicated greater disturbance of the ability being tested.

When age and sex were taken into account (Tables 5.13 to 5.16) Group 1 again showed less impairment than the other patient groups. Overall, patients were different from controls in showing greater disturbance of abilities tested. Within the patient groups a general severity effect was evident, such that Group 1 showed less impairment than Group 2, Group 2 less than Group 3, and Group three was similar to Group 4. The differences between patient groups did not reveal any clear pattern of neuropsychological dysfunction, but rather were heterogeneously distributed among cognitive, motor, and sensory-perceptual variables.

5.4.6 Results of Hypotheses

5 At the end of chapter 4, seven hypotheses were presented in the null form. These are restated below:

1. **There are no differences between psychiatric patients and controls, as assessed by a battery of neuropsychological tests.**

   Results of MANOVA with follow-up Scheffe contrasts shows that patients are different from controls on all neuropsychological variables (see Tables 5.12 to 5.16). Hypothesis 1 is rejected.

2. **There are no differences between unselected non-hallucinating patients (Group 1), unselected hallucinating patients (Group 2), selected non-hallucinating patients (Group 3), and selected hallucinating patients (Group 4), as assessed by a battery of neuropsychological tests.**

   Results of MANOVA comparing Groups 1, 2, 3, and 4 across all neuropsychological variables shows group membership to be significant at $p<.000$ (see Table 5.12). Hypothesis 2 is rejected.

3. **There are no differences between unselected and selected psychiatric patients, as assessed by a battery of neuropsychological tests.**

   Results of MANOVA comparing Groups 1 and 2, the unselected patients, against Group 3 and 4, the selected patients, across all neuropsychological variables shows a group effect at the $p<.000$ level of significance. Hypothesis 3 is rejected.

4. **There are no differences between unselected non-hallucinating patients and selected non-hallucinating patients, as assessed by a battery of neuropsychological tests.**

Results of MANOVA comparing Group 1, the unselected non-hallucinators, and Group 3, the selected non-hallucinators, across all neuropsychological variables shows a group effect at the $p<.000$ level of significance. Hypothesis 4 is reject.

5. **There are no differences between unselected hallucinating patients and selected hallucinating patients, as assessed by a battery of neuropsychological tests.**

Results of MANOVA comparing Group 2, the unselected hallucinating patients, and Group 4, the selected hallucinating patients, across all neuropsychological variables shows a group effect at the $p<.002$ level of significance. Hypothesis 5 is rejected.

6. **There are no differences between unselected non-hallucinating patients and unselected hallucinating patients, as assessed by a battery of neuropsychological tests.**

Results of MANOVA comparing Group 1, the unselected non-hallucinating patients, with Group 2, the unselected hallucinating patients, across all neuropsychological variables shows a group effect at the $p<.000$ level of significant. Hypothesis 6 is rejected.

7. **There are no differences between selected non-hallucinating patients and selected hallucinating patients, as assessed by a battery of neuropsychological tests.**

Results of MANOVA comparing Group 3, the selected non-hallucinating patients, with Group 4, the selected hallucinated patients, across all neuropsychological variables does not show a significant group effect. Hypothesis 7 is accepted.

Table 5.17 summarizes the MANOVA results by group membership for to hypotheses 1 through 7. With significant F values indicating rejection of the null hypotheses in six out of seven cases.

5.5 Supplementary Analyses

Following the main data analysis, several additional procedures were carried out. These included an examination of the discriminatory ability of ratio scores constructed from the neuropsychological tests used in the investigation, a correlation analysis, and a separate factor analysis of the patient and control samples.

5.5.1 Ratio Scores

In order to more fully examine the effect of psychiatric symptomatology on neuropsychological test scores, ratio scores based on tests by brain region were developed. The idea was to compare combinations of test

Table 5.17

Multivarate Analysis of Variance of Main Effects for Group Membership as Described in Hypotheses 1 to 7

Hypotheses	Test Name	Value	F	Hypoth DF	Error DF	Significance of F
1	Wilks Lambda	.19	4.30	140.0	1149.4	.000
2	Wilks Lambda	.18	2.43	105.0	333.0	.000
3	Wilks Lambda	.44	4.35	35.0	119.0	.000
4	Wilks Lambda	.25	3.71	35.0	44.4	.000
5	Wilks Lambda	.26	2.74	35.0	33.0	.002
6	Wilks Lambda	.37	3.18	35.0	65.0	.000
7	Wilks Lambda	.26	.99	35.0	12.0	.536

scores known to have general sensitivity to certain brain areas. Eleven rank ratios were derived using tests thought to be differentially sensitive to the left and right hemisphere and to the anterior/posterior dimension. Scores were setup as a ratio of left to right and Scheffe comparisons used to simultaneously test all five groups on the ratio score. The eleven rank ratios, from front to back of the brain were: Wisconsin Card Sorting Test - Errors over Halstead Category Test - Errors, Wisconsin Card Sorting Test - Categories over Halstead Category Test - Errors, Purdue Pegboard - Preferred Hand over Purdue Pegboard - Nonpreferred Hand, Trail Making Test - B over Trail Making Test A, Dynamometer - Preferred Hand over Dynamometer - Nonpreferred Hand, Finger Tapping - Preferred Hand over Finger Tapping - Nonpreferred Hand, Speech Sounds Perception Test over Seashore Rhythm Test, the average rank of Information, Comprehension, Arithmetic, Similarities, Digit-Span, and Vocabulary over the average rank of Digit Symbol, Picture Completion, Block Design, Picture Arrangement, and Object Assembly. In addition the following rank ratios were tested, Finger Localization - Preferred Hand - Single Stimulation over Finger Localization - Nonpreferred Hand - Single Stimulation, Finger Localization - Preferred Hand - Double Stimulation over Finger Localization - Nonpreferred Hand - Double Stimulation, and L.J. Tactile Recognition - Preferred Hand over L.J. Tactile Recognition -

Nonpreferred Hand.

The rank ratio scores did not prove to be sensitive to the presence or absence of hallucinations among the patient groups. None of the Scheffe contrasts reached significant at the p<.05 level. This may have been, in part, due to the psychometric unreliability of ratio or difference scores (Clark, 1986; Dunlap & Kemery, 1988; Payne, 1958; Payne & Jones, 1957).

5.5.2 Correlational Analyses

Spearman Rho correlation coefficients were computed for controls and patients separately. It should be noted, that for ranks, the Spearman Rho statistic is essentially equivalent to the Pearson product moment correlation (Fowler, 1987). Neuropsychological test variables were correlated with WAIS-R subtests and with Verbal, Performance, and Full Scale IQ. Results are given in Appendix 1. Inspection of Table AI.1 the correlation matrix for controls (n=182) shows minimal correlation between neuropsychological and intellectual variables. Of the 336 correlations computed only 9 (2.5%) are greater than or equal to .30. The single highest correlation is between the Halstead Category Test and Full Scale IQ (.40). These values are consistent with the published normative studies of Yeudall et al. (1986, 1987).

By contrast correlations among the patient sample (n=161) are in the .30 to .50 range (see Table AI.2). The single highest correlation is .60, between Trail Making -

Part B and Full Scale IQ and of the 336 correlations computed 129 (38.4%) are greater than or equal to .30. Thus it is clear that in this sample intellectual factors have an impact on neuropsychological test performance. However, the shared variance between the two sets of measures is on average 10 to 25 percent, which leaves a substantial amount of test variance unaccounted for.

In addition to nonparametric Spearman Rho correlations, canonical correlations between the Wechsler scales and neuropsychological variables was also performed. Canonical correlation is a technique whereby the degree of association between a linear or weighted set of criterion variables and a linear or weighted set of predictor variables may be estimated (Isaac & Michael, 1981). Correlations were computed separately for patients and controls. Tables AI.3 and AI.4 present the canonical correlations for controls and patients as well as results of the canonical redundancy indices. Table AI.5 presents squared multiple correlations for predicting WAIS-R variables from remaining WAIS-R variables and neuropsychological variables. While Table AI.6 gives the squared multiple correlations for predicting neuropsychological variables from the WAIS-R and remaining neuropsychological variables. Results of the redundancy index indicates that for patients 28% of the WAIS-R may be predicted from the neuropsychological variables, whereas only 18% of the neuropsychological test variables variance can be predicted from the WAIS-R. For controls 10% of WAIS-R variance can be predicted by the neuropsychological test scores, and 4% of neuropsychological test score variance is predictable from the WAIS-R. In the combined sample of patients and controls, 34.5% of the WAIS-R variance can be predicted from neuropsychological variables, and 23.5% of the neuropsychological test scores variance can be predicted by the WAIS-R.

5.5.3 Factor Analysis

In the present study the number of factors was determined primarily by interpretability, as well as by use of the scree test (Cattell, 1966), Velicer's minimal average partial (Reddon, 1985a; 1985b; Velicer, 1976), and a permutation procedure developed by Reddon (1984). For interpretive purposes only items with a loading of $\geq .30$ were considered. Principal components analysis with varimax rotation was used to develop a 7 factor solution for patients and controls. These factors accounted for 66.8% and 51.5% of the total variance respectively. Determination of the number of factors is problematic in that no definitive criterion for the number of factors to extract has yet been developed (Franzen & Golden, 1983; Hill, Reddon, & Jackson, 1985; Reddon, 1984; Zwick & Velicer, 1986).

In order to ascertain the degree of similarity between the two factor patterns, an orthogonal procrustean transformation was applied to the data (Schönemann, 1966).

This used the seven factor patient solution as the target matrix and coefficients of congruence were computed (Mulaik, 1972). Recently, Broadbooks and Elmore (1987) have shown this statistic to be conservative for values grater than .50 and thus an underestimate of the actual population value. The following values were obtained by rotating control factors into patient factors; .90, .83, .87, .87, .88, .79, and .53. It can be seen that with the exception of the last factor, a high degree of similarity exists between the factor structure of the two samples.

Following is a description of the factor structure for the patient group. Where control factors differ, they will be discussed separately. Table 5.18 lists the abbreviations for variable names, while Tables 5.19 and 5.20 present the sorted Varimax rotated principal components and communalities for controls and patients. Table 5.21 and 5.22 list variables with a loading of $\geq$3 that define the factors for controls and patients. The unrotated Spearman Rho correlation matrices for controls and patients are reported in Tables 5.23 and 5.24.

Factor 1. This factor is defined by nine variables with a salience of $\geq$.3 (see Table 5.22 and compare with Table 5.21). It is interpreted as a verbal comprehension and memory factor. The first five defining variables are all from the verbal portion of the WAIS-R. Measures of verbal fluency, recall of numbers, graphomotor speed, and logical interpretation of social situations also load on this factor. The last two variables, Digit Symbol and Picture Arrangement from the WAIS-R have minimal loadings on the factor, however, it is noted that both tasks employ some degree of verbal mediation in their solution. Neuropsychologically this factor represents primarily dominant hemisphere functioning, especially of the frontal and temporal lobe regions.

Factor II. Eight variables load significantly on this factor which involves primarily tactile recognition and attention. The first four variables require cross-modal identification of sensory stimulation, while the Seashore Rhythm Test and Speech Sounds Perception Test require non-tactile pattern recognition. Digit-Span and Oral Word Fluency require sustained attention, as do the two previously mentioned tasks. Overall, the factor is defined by the sensory-perceptual processes required by the Finger-Localization Test. But a strong attentional component is also evident. Neuropsychologically this factor is complex, involving frontally and subcortically mediated attentional mechanisms as well as the pattern recognition and cross-modal matching systems of the parietal lobes.

Factor III. The third factor is defined by thirteen variables, reflecting primarily visuospatial processing. The visual, tactile and auditory modalities are all involved, with visual processing emphasized in 9 of the 13 variables. The high loading of the Speech Sounds

Perception Test (.57) is surprising. But as well as auditory processing the task also requires visual graphomotor abilities, reading and underlining the spatial location of the answer. Because of the range and complexity of tasks identified in this factor, it it interpreted neuropsychologically as a general right hemisphere factor, possibly more temporoparietal than frontotemporal in nature.

Factor IV. This factor is defined by seven variables, six of which involve rapid motor response. The Purdue-Pegboard Test is the main determinant of this factor and this task requires fine motor coordination and motor speed. Other variables loading on the factor also have a significant motor output component (Oral Word Fluency, Digit-Symbol). The Finger Localization test (Preferred Hand-Single Stimulation) also loads minimally on this perceptual-motor speed factor. This may be due to the motor response components of this task, pointing and naming aloud. It is also noted that these complex motor tasks have cognitive components as well as motor expression elements. Neuropsychologically, this factor is thought to reflect the functioning of the premotor regions of the frontal lobes.

Factor V. This factor is defined by four variables which reflect preferred and nonpreferred hand performance on tests of grip strength and finger tapping speed. The tasks are less cognitively complex than those defining factor

IV, and they require, relatively speaking, more gross motor skill. From a neuropsychological perspective this factor involves the function of the posterior frontal motor systems, that is primary motor cortex.

Factor VI. Seven variables define this factor. It is primarily determined by a complex tactile recognition and matching task. The other four variables that load on this factor all involve pattern recognition, have a motor component, and all require rapid responding. The crucial elements in the factor are tactile pattern recognition and speed of response. Neuropsychologically the factor probably reflects parietal lobe functioning, with the additional tests loading on the factor suggesting a nondominant hemisphere emphasis (Object Assembly, Block Design, Digit Symbol, and Trial Making Test - Part A).

Factor VII. The last factor is defined by seven variables. The most significant of these (Wisconsin Card Sorting Test, Halstead Category Test, Trail Making Test - part B) all require conceptual flexibility and abstract reasoning ability. The other variables have much lower loadings and involve perceptual matching, sustained attention and visual scanning. This factor is defined primarily in terms of cognitive processes, rather than in terms of motor or sensory-perceptual variables. In terms of cerebral processes, this factor involves anterior frontal lobe functions, that is the prefrontal lobes.

There is a substantial overlap between the factors identified on patients and on controls. Using the patients as a reference, factor I goes with factor II (controls), factor II goes with factor V (controls), factor III goes with factor VII (controls), factor IV goes with factor IV (controls), factor V goes with factor I (controls), factor VI goes with factor III (controls), and factor VII goes with factor VI (controls). Comparing patient factors with control factors in terms of the defining variables, good agreement is present for factors I/II, II/V, IV/IV, V/I and VI/III. Less agreement is evident on the III/VII and VII/VI. Factors VI and VII for controls are described below.

Factor VI: Controls For the control sample, eight variables determine this factor. Loadings range from .31 to .62, with verbally mediated cognitive tasks defining the factor. Additional measures involve less complex cognitive functions, and have a reduced language component. The main difference between the patient factor (VII) and the analogous control factor (VI) is that the Halstead Category Test does not load on this factor. This verbal concept formation factor in controls is seen as reflecting prefrontal cognitive activity, (possibly dominant > nondominant hemisphere).

Factor VII: Controls. Six variables define this factor, four of which require visuospatial concept formation. The Arithmetic and Information subtest loadings on this factor may be due to visualization elements in these tasks.

Comparison with the analogue patient factor (III) shows the control factor to be a less general nondominant hemisphere measure. Neuropsychologically it is hypothesized to be a nondominant temporoparietal factor.

Table 5.18

Abbreviations Used to Describe Variable Names in Principal Component Analyses of Patient and Control Data

Abbreviation	Variable Name
CPM	Colored Progressive Matrices
OWF	Oral Word Fluency
PURDP	Purdue Pegboard, Preferred Hand
PURDNP	Purdue Pegboard, Nonpreferred Hand
PURDB	Purdue Pegboard, Both Hands
PURDA	Purdue Pegboard, Assemblies
LJPT	L.J. Tactile Recognition, Preferred Hand Time
LJNPT	L.J. Tactile Recognition, Nonpreferred Hand Time
LJBT	L.J. Tactile Recognition, Both Hands Time
WCST	Wisconsin Card Sorting Test, Errors
WCSCAT	Wisconsin Card Sorting Test, Categories
SPEECHS	Speech Sounds Perception Test
TRAILA	Trail Making Test, Part A
TRAILB	Trail Making Test, Part B
HALCAT	Halstead Category Test, Errors

Table 5.18 (Continued)

Abbreviation	Variable Name
FTAPP	Finger Tapping Test, Preferred Hand
FTAPNP	Finger Tapping Test, Nonpreferred Hand
DYNP	Dynamometer, Preferred Hand
DYNNP	Dynamometer, Nonpreferred Hand
SEAR	Seashore Rhythm Test
FLOCPSS	Finger Localization, Preferred Hand, Single Stimulation
FLOPDS	Finger Localization, Preferred Hand, Double Stimulation
FLOCNPSS	Finger Localization, Nonpreferred Hand, Single Stimulation
FLOCNPDS	Finger Localization, Nonpreferred Hand, Double Stimulation
INFO	WAIS-R Information
COMP	WAIS-R Comprehension
ARIT	WAIS-R Arithmetic
SIMI	WAIS-R Similarities
DSPA	WAIS-R Digit Span
VOCA	WAIS-R Vocabulary
DSYM	WAIS-R Digit Symbol
PCOM	WAIS-R Picture Completion
BDES	WAIS-R Block Design
PARR	WAIS-R Picture Arrangement
OASS	WAIS-R Object Assembly

Table 5.19

Sorted Varimax Rotated Principal Component Loadings for Controls (n=182), 7 Factor Solution[a]

	Factor							
Variable	I	II	III	IV	V	VI	VII	h^2
DYNP	.85	.04	.01	-.26	.04	-.03	.18	.83
DYNNP	.83	.03	-.06	-.23	-.02	.00	.16	.78
FTAPP	.77	.27	.20	.08	.05	.05	-.20	.76
FTAPNP	.74	.26	.12	.07	.07	.10	-.29	.73
VOCA	.15	.80	-.04	-.06	.07	.05	.11	.67
COMP	.10	.67	.09	.13	-.08	-.05	.00	.49
INFO	.23	.67	-.09	-.15	-.08	-.03	.32	.64
SIMI	.01	.55	.00	.00	-.09	.25	.09	.40
ARIT	.16	.42	.03	-.19	.10	.31	.34	.45
OWF	.13	.34	-.25	.21	.06	-.03	.18	.27
DSPA	.05	.29	-.02	-.05	.23	.28	.11	.23
LJPT	-.01	.05	.85	.04	.00	-.04	.10	.73
LJNPT	.11	-.02	.84	.02	-.07	.06	.10	.74
LJBT	.09	-.07	.84	-.02	-.05	.13	.09	.74
PCOM	.32	.30	.36	.00	.04	.00	.21	.37
PURDP	-.13	.00	.05	.79	-.04	-.04	.04	.65
PURDB	-.05	-.02	-.07	.77	-.03	.13	.19	.66
PURDP	-.04	.06	-.05	.76	-.02	.01	-.08	.60
PURDA	-.05	-.03	.19	.58	.25	.26	-.02	.51
FLOCNPDS	.01	.04	-.07	.11	.75	.16	-.03	.01
FLOCPDS	.10	-.03	-.03	.01	.72	.16	.09	.56
FLOCPSS	-.01	-.03	-.08	-.04	.62	-.24	.22	.49
FLOCNPSS	.01	-.10	-.01	-.10	.57	.19	.04	.38
SEAR	-.23	.28	.12	.19	.39	.10	-.27	.41
SPEECHS	.26	.26	.15	.15	.33	-.18	-.03	.32

Table 5.19 (continued)

	Factor							
Variable	I	II	III	IV	V	VI	VII	h^2
WCST	.04	.27	-.03	.09	.11	.62	-.07	.49
WCSCAT	.03	.07	.08	-.02	.02	.58	.27	.36
TRAILB	.01	-.08	.14	.22	.10	.53	.27	.44
DSYM	-.05	-.09	-.04	.26	.12	.49	-.10	.34
TRAILA	.28	-.23	.30	.22	.07	.37	.19	.45
CPM	-.04	.09	-.02	-.08	-.02	.36	.32	.24
PARR	-.07	.29	.25	-.22	.11	.33	-.12	.33
BDES	.04	.16	.12	.09	.10	.15	.63	.48
OASS	-.05	.14	.22	.06	.03	-.06	.59	.43
HALCAT	-.01	.34	.06	.08	.26	.11	.47	.43
	(30.4)	(8.6)	(8.2)	(6.3)	(5.5)	(4.4)	(3.5)[b]	

[a] This solution accounts for 51.5% of the total variance.

[b] Values in brackets refer to the percentage of variance accounted for by each factor.

Table 5.20

Sorted Varimax Rotated Principal Component Loadings

for Patients (n=161), 7 Factor Solution[a]

	Factor							
Variable	I	II	III	IV	V	VI	VII	h^2
INFO	.83	.05	.05	.06	.12	.00	.09	.72
VOCA	.81	.21	.26	.09	-.01	-.07	.08	.78
COMP	.76	.11	.23	.15	.07	.05	.03	.70
ARIT	.73	.28	-.02	.14	.21	.03	.21	.72
SIMI	.65	.11	.40	.09	.06	.03	.06	.61
FLOCNPDS	.13	.77	-.04	.06	.14	.10	.33	.75
FLOCNPSS	.11	.76	-.01	.14	.07	.09	-.05	.62
FLOCPDS	.08	.74	.03	.10	.18	.22	.29	.73
FLOCPSS	.05	.61	.19	.32	.04	.04	-.03	.52
DSPA	.34	.60	.18	.01	-.02	-.03	.23	.56
SEAR	.25	.53	.44	.08	.03	.06	-.03	.55
BDES	.22	.04	.63	.12	.22	.37	.12	.67
OASS	.08	-.05	.62	.00	.12	.38	.06	.55
PARR	.30	-.14	.57	.05	.27	.09	.24	.58
SPEECHS	.12	.46	.57	.10	.02	-.09	.12	.58
PCOM	.26	.21	.55	.03	.27	.11	.30	.59
CPM	.29	.21	.51	.04	.02	-.07	.22	.45
TRAILA	.04	.12	.40	.25	.23	.32	.39	.55
PURDB	.14	.07	-.06	.87	.05	.09	.19	.84
PURDP	.08	.13	.07	.84	.02	.01	.07	.73
PURDNP	.08	.11	.04	.83	.11	.19	.10	.75
PURDA	.08	.22	.31	.62	-.01	.28	.22	.66
OWF	.37	.40	.12	.46	-.01	.10	.01	.53
DSYM	.33	.24	.35	.35	-.09	.32	.26	.60
DYNNP	.06	-.04	.01	-.04	.89	.21	.04	.85
DYNP	.09	-.03	.08	-.03	.89	.18	.02	.84
FTAPNP	.04	.26	.24	.15	.72	-.07	.03	.67
FTAPP	.18	.27	.24	.12	.64	-.10	-.03	.60

Table 5.20 (continued)

Variable	Factor I	II	III	IV	V	VI	VII	h^2
LJNPT	.00	.02	.12	.08	.01	.89	.05	.82
LJPT	-.05	.11	.09	.13	.05	.83	.05	.74
LJBT	.02	.16	.10	.13	.16	.79	.20	.75
WCST	.10	.17	.17	.06	-.06	.07	.82	.76
WCSCAT	.06	.01	.07	.26	-.04	.09	.79	.72
HALCAT	.22	.22	.38	.08	.20	.12	.60	.67
TRAILB	.25	.25	.32	.26	.12	.25	.52	.65
	(13.6)	(9.8)	(7.7)	(6.2)	(5.8)	(4.3)	(4.0)[b]	

[a] This solution accounts for 66.8% of the total variance.

[b] Values in brackets refer to the percentage of variance accounted for by each factor.

Table 5.21

Controls, 7 Factor Solution, 51.5% of Total Variance

Factor I		Factor II		Factor III		Factor IV		Factor V		Factor VI		Factor VII	
DYNP	.85	VOCAB	.80	LJPT	.85	PURDP	.79	FLOCNPDS	.75	WCST	.62	BDES	.63
DYNNP	.83	COMP	.67	LJNPT	.84	PURDB	.77	FLOCPDS	.72	WCSCAT	.58	OASS	.59
FTAPP	.77	INFO	.67	LJBT	.84	PURDNP	.76	FLOCPSS	.62	TRAILB	.53	HALCAT	.47
FTAPNP	.74	SIMI	.55	PCOM	.36	PURDA	.58	FLOCNPSS	.57	DSYM	.49	ARIT	.34
PCOM	.32	ARIT	.42	TRAILA	.30	(6.2%)		SEAR	.39	TRAILA	.37	INFO	.32
(13.6%)		OWF	.34	(7.7%)				SPEECHS	.33	CPM	.36	CPM	.32
		HALCAT	.34					(5.8%)		PARR	.33	(4.0%)	
		PCOM	.30							ARIT	.31		
		(9.8%)								(4.3%)			

Note. Percentages in parentheses refer to variance accounted for by each factor.

Table 5.22

Patients, 7 Factor Solution, 66.8% of Total Variance

Factor I		Factor II		Factor III		Factor IV		Factor V		Factor VI		Factor VII	
INFO	.83	FLOCNPDS	.77	BDES	.63	PURDB	.87	DYNP	.89	LJNPT	.89	WCST	.82
VOCAB	.81	FLOCNPSS	.76	OASS	.62	PURDP	.84	DYNNP	.89	LJPT	.83	WCSCAT	.79
COMP	.76	FLOCPDS	.74	PARR	.57	PURDNP	.83	FTAPNP	.72	LJBT	.79	HALCAT	.60
ARITH	.73	FLOCPSS	.61	SPEECHS	.57	PURDA	.62	FTAPP	.64	OASS	.38	TRAILB	.52
SIMI	.65	DSPA	.60	PCOM	.55	OWF	.46	(5.5%)		BDES	.37	TRAILA	.39
OWF	.37	SEAR	.53	CPM	.51	DSYM	.35			DSYM	.32	FLOCNPDS	.33
DSPA	.34	SPEECHS	.46	SEAR	.44	FLOCPSS	.32			TRAILA	.32	PCOM	.30
DSYM	.33	OWF	.40	SIMI	.40	(6.3%)				(4.4%)		(3.5%)	
PARR	.30	(8.6%)		TRAILA	.40								
(30.4%)				HALCAT	.38								
				DSYM	.35								
				TRAILB	.32								
				PURDA	.31								
				(8.2%)									

Note. Percentages in parentheses refer to variance accounted for by each factor.

Table 5.23

Spearman Rank Correlation Matrix (Lower Triangular) for Controls (N=182)

CPM	1.00												
OWF	.01	1.00											
PURDP	.00	.07	1.00										
PURDNP	-.05	.07	.53	1.00									
PURDB	-.01	.14	.50	.55	1.00								
PURDA	.12	.02	.37	.34	.40	1.00							
LJPT	.01	-.08	.04	.02	.01	.10	1.00						
LJNPT	-.02	-.07	.02	-.03	.00	.06	.71	1.00					
LJBT	.07	-.11	-.02	-.12	-.03	.13	.62	.71	1.00				
WCST	.16	.15	.00	.15	.08	.14	-.04	.03	.05	1.00			
WCSCAT	.18	.07	-.03	.07	.06	.08	.10	.15	.17	.39	1.00		
SPEECHS	.00	.04	.12	.07	-.05	.03	.06	.02	.02	.08	.06	1.00	
TRAILA	.04	-.01	.17	-.02	.17	.20	.19	.21	.29	.10	.14	.07	1.00
TRAILB	.11	.06	.13	.08	.18	.27	.13	.13	.14	.16	.17	.01	.46
	1.00												
HALCAT	.17	.12	.00	.07	.12	.15	.12	-.01	.05	.18	.14	.16	.08
	.18	1.00											
FTAPP	.00	.05	-.06	-.03	-.01	.10	.10	.17	.19	.17	.07	.29	.20
	-.06	.10	1.00										
FTAPNP	-.03	.10	-.08	.01	-.04	.06	.04	.13	.12	.19	.07	.20	.11
	-.03	-.05	.80	1.00									
DYNP	.03	.11	-.29	-.20	-.22	-.21	.05	.16	.10	.03	.06	.17	.16
	.02	.09	.52	.45	1.00								
DYNNP	.05	.14	-.27	-.13	-.20	-.21	.00	.09	.06	.06	.09	.15	.10
	.00	.05	.45	.45	.91	1.00							
SEAR	-.01	.05	.17	.15	.08	.17	.12	-.03	-.01	.12	.08	.16	.06
	.11	.08	-.02	.00	-.14	-.17	1.00						
FLOCPSS	.01	.12	-.04	-.07	.00	.10	-.10	-.10	-.07	-.02	-.01	.09	.03
	-.02	.13	-.06	-.06	.08	.03	.08	1.00					
FLOCPDS	.08	.10	-.06	.02	.05	.25	.00	.02	-.02	.17	.14	.14	.11
	.18	.20	.06	.10	.08	.06	.15	.31	1.00				
FLOCNPSS	.05	.02	-.06	-.03	-.06	.13	-.02	-.03	.01	.16	.10	.05	.07
	.11	.05	.00	.04	.05	.01	.05	.26	.29	1.00			
FLOCNPDS	.01	.08	.02	.05	.15	.22	-.03	-.09	-.08	.19	.04	.16	.07
	.12	.23	.07	.08	.00	-.05	.23	.26	.49	.35	1.00		
INFO	.13	.22	-.12	-.05	-.03	-.18	-.03	.05	-.11	.08	.00	.16	-.02
	.05	.28	.24	.21	.30	.24	-.07	-.01	.04	-.07	.02	1.00	
COMP	.03	.31	.05	.09	.08	.09	.07	.08	.05	.21	.08	.12	-.04
	.00	.13	.22	.18	.10	.11	.02	.09	-.08	-.01	-.11	.41	1.00

Table 5.23 (Continued)

ARIT	.19	.09	-.07	-.03	-.03	-.01	.11	.12	.07	.16	.18	.05	.01
	.17	.28	.11	.13	.26	.27	.06	.01	.16	.12	.10	.42	.15
	1.00												
SIMI	.06	.17	-.01	-.08	.05	.04	.07	.00	.02	.21	.13	.03	.05
	.11	.23	.19	.17	.03	.03	.09	-.07	-.02	.01	.04	.35	.31
	.21	1.00											
DSPA	.09	.13	.01	-.03	.01	.05	.01	-.01	.04	.10	.16	.11	.14
	.19	.23	.07	.13	.12	.10	.33	.05	.14	.08	.14	.17	.05
	.37	.12	1.00										
VOCA	.15	.28	-.03	-.01	-.01	-.07	.01	.00	.00	.20	.08	.21	-.06
	.01	.28	.26	.24	.20	.19	.13	.03	.07	.01	.10	.61	.45
	.40	.38	.28	1.00									
DSYM	.06	.08	.20	.10	.21	.27	-.07	.04	.02	.19	.08	.02	.22
	.25	-.06	.04	.05	-.17	-.12	.12	-.04	.08	.11	.24	-.04	-.08
	.04	.09	.08	-.01	1.00								
PCOM	.11	-.04	-.05	-.03	-.02	-.02	.26	.20	.27	.10	.07	.24	.20
	.16	.22	.40	.33	.24	.18	-.04	.01	.06	-.02	.02	.29	.17
	.24	.14	.11	.27	-.07	1.00							
BDES	.13	.12	.02	.00	.15	.13	.16	.14	.13	.16	.19	.12	.14
	.21	.34	.08	-.01	.10	.07	-.01	.07	.10	.04	.09	.22	.11
	.22	.19	.25	.18	.09	.21	1.00						
PARR	.11	-.08	-.10	-.04	-.10	.02	.10	.13	.19	.14	.09	.00	.08
	.14	.13	.09	.08	.01	.01	.07	.06	.07	.08	.07	.17	.20
	.28	.05	.09	.21	.08	.18	.06	1.00					
OASS	.11	.09	.06	-.06	.13	.04	.19	.25	.19	.08	.12	.08	.08
	.03	.17	.03	.03	.00	.02	.03	.12	.03	.09	-.02	.22	.11
	.22	.15	.04	.10	.04	.16	.38	-.04	1.00				

Note: The format of the correlation matrix is in lower triangular form with up to 13 elements per row. With 35 variables 1 to 13 contain rows1 to 13 up to and including the diagnonal. Line 14 contains the first 13 elements of the 14th row of the correlation matrix. Line 15 contains the disgonal element of row 14 of the correlation matrix. Line 16 contains the first 13 elements of row 15, and line 17 contains the next 2elements of row 15 of the correlation matrix, and so on.

Note: Critical values for 1-tail significance test of Spearman's Rho, are: p=.10, rho=.10; p=.05, rho=.12; p=.01, rho=1.7; p=.001, rho=.23.

Table 5.24

Spearman Rank Correlation Matrix (Lower Triangular) for Patients (N=161)

CPM	1.00												
OWF	.19	1.00											
PURDP	.28	.49	1.00										
PURDNP	.13	.38	.66	1.00									
PURDB	.15	.50	.74	.74	1.00								
PURDA	.23	.46	.55	.54	.63	1.00							
LJPT	.07	.22	.17	.22	.26	.39	1.0C						
LJNPT	.01	.25	.14	.18	.18	.33	.71	1.00					
LJBT	.12	.27	.22	.30	.26	.40	.66	.71	1.00				
WCST	.26	.26	.20	.18	.23	.37	.15	.16	.25	1.00			
WCSCAT	.24	.19	.28	.28	.38	.37	.18	.19	.26	.70	1.00		
SPEECHS	.38	.38	.31	.24	.21	.37	.07	.06	.15	.23	.20	1.00	
TRAILA	.28	.32	.37	.37	.35	.52	.38	.37	.41	.40	.34	.35	1.00
TRAILB	.36	.37	.36	.43	.44	.58	.31	.28	.43	.49	.49	.47	.65
	1.00												
HALCAT	.48	.35	.29	.26	.30	.41	.21	.25	.40	.56	.48	.42	.46
	.59	1.00											
FTAPP	.23	.31	.14	.23	.14	.26	.12	.02	.15	.07	.04	.30	.25
	.25	.36	1.00										
FTAPNP	.22	.30	.22	.24	.19	.25	.13	.07	.21	.15	.14	.28	.36
	.28	.34	.75	1.00									
DYNP	.12	.04	.10	.11	.07	.10	.14	.16	.27	.03	.02	.10	.29
	.25	.23	.41	.48	1.00								
DYNNP	.05	.03	.06	.12	.07	.09	.15	.18	.28	.03	.00	.07	.30
	.25	.21	.38	.47	.94	1.00							
SEAR	.38	.39	.25	.23	.26	.37	.20	.10	.20	.24	.14	.50	.31
	.43	.33	.26	.33	.16	.13	1.00						
FLOCPSS	.23	.37	.37	.34	.28	.35	.15	.07	.24	.18	.18	.35	.25
	.33	.27	.23	.27	.07	.01	.36	1.00					
FLOCPDS	.25	.39	.25	.29	.26	.37	.28	.24	.37	.36	.27	.34	.35
	.50	.47	.30	.35	.17	.18	.43	.51	1.00				
FLOCNPSS	.20	.35	.24	.26	.21	.27	.11	.10	.18	.12	.04	.30	.16
	.30	.30	.22	.23	.10	.06	.42	.53	.59	1.00			
FLOCNPDS	.24	.35	.31	.23	.28	.36	.20	.11	.29	.39	.29	.39	.31
	.44	.47	.24	.29	.14	.14	.47	.49	.73	.60	1.00		
INFO	.34	.30	.20	.18	.21	.11	.04	-.04	.06	.11	.16	.17	.13
	.32	.30	.25	.19	.14	.09	.31	.17	.19	.19	.22	1.00	
COMP	.37	.47	.32	.24	.25	.40	.04	.12	.13	.23	.18	.36	.32
	.43	.30	.28	.23	.19	.16	.46	.23	.25	.20	.25	.57	1.00

Table 5.24 (Continued)

ARIT	.35	.41	.32	.28	.31	.37	.08	.05	.20	.28	.24	.35	.34
	.52	.37	.36	.30	.24	.24	.38	.29	.40	.34	.43	.56	.66
	1.00												
SIMI	.42	.36	.19	.23	.19	.25	.03	.08	.16	.19	.24	.39	.23
	.39	.33	.24	.21	.15	.11	.38	.32	.25	.19	.24	.62	.63
	.49	1.00											
DSPA	.31	.41	.23	.22	.22	.26	.05	.02	.16	.27	.20	.50	.27
	.42	.40	.19	.17	.04	.01	.47	.37	.43	.42	.49	.28	.36
	.48	.36	1.00										
VOCA	.44	.53	.26	.17	.25	.29	.04	.03	.08	.27	.19	.37	.24
	.39	.35	.31	.24	.05	.01	.49	.29	.27	.28	.31	.72	.71
	.62	.63	.45	1.00									
DSYM	.27	.41	.35	.37	.38	.58	.31	.30	.34	.37	.30	.31	.48
	.59	.42	.20	.16	.10	.08	.35	.31	.41	.34	.36	.30	.49
	.54	.40	.39	.43	1.00								
PCOM	.36	.29	.19	.16	.12	.42	.18	.18	.28	.40	.28	.38	.46
	.49	.47	.33	.40	.33	.30	.43	.24	.37	.20	.32	.29	.45
	.39	.38	.31	.43	.49	1.00							
BDES	.38	.29	.25	.25	.25	.38	.29	.32	.40	.26	.23	.36	.44
	.54	.44	.29	.36	.36	.31	.37	.23	.28	.17	.25	.35	.39
	.36	.48	.24	.35	.48	.54	1.00						
PARR	.32	.12	.23	.19	.15	.36	.14	.12	.18	.30	.22	.29	.43
	.39	.43	.32	.30	.32	.26	.21	.15	.16	.08	.15	.32	.42
	.34	.46	.17	.36	.37	.59	.56	1.00					
OASS	.26	.10	.13	.11	.07	.18	.29	.36	.30	.21	.25	.28	.34
	.28	.25	.17	.24	.30	.23	.29	.17	.17	.07	.12	.19	.26
	.15	.39	.08	.20	.30	.35	.56	.33	1.00				

Note: Critical values for 1-tail significance test of Spearman's Rho, are: p=.10, rho=.10; p=.05, rho=.13; p=.01, rho=.18; p=.001, rho=.24

Chapter VI

Discussion

6.1 Introduction

This final chapter consists of three sections: a discussion of the findings and limitations of the investigation, comments on the theoretical implications of the findings, and lastly some suggested directions for future research.

6.2 Findings and Limitations of the Investigation

Five groups of subjects were assessed in this study; unselected non-hallucinators (n=52), unselected hallucinators (n=55), selected non-hallucinators (n=34), selected hallucinators (n=20), and controls (n=182). Chi-squared statistics showed no effects for group membership by sex, handedness, or age. It can be seen from table 5.1 that IQ scores for control subjects were significantly higher than for patient groups. A not unreasonable finding in view of their lack of brain dysfunction. Analysis of variance showed significant main effects for sex, age, and group membership for Verbal, Performance, and Full Scale IQ (see tables 5.9, 5.10 and 5.11). In order to enhance sensitivity from a neuropsychological perspective, subsequent analyses used Wechsler subtest scores rather than composite IQ scores.

Multivariate analysis of variance showed effects for age, sex, and group membership (see table 5.12). Scheffe contrasts were performed separately on younger females, older females, younger males, and older males. In each case controls were clearly differentiated from all patient groups, reflecting the sensitivity of neuropsychological and cognitive variables (see Tables 5.12 to 5.16). Based on the rank order of group means a severity dimension emerged (see table 5.12). Unselected non-hallucinators were less impaired than unselected hallucinators, who in turn were less impaired than selected non-hallucinators. The selected non-hallucinators showed a similar level of impairment to the selected hallucinators.

In terms of the seven hypotheses stated in Chapter IV, six were rejected. These hypotheses were designed to compare selected hallucinators and selected non-hallucinators and also to compare unselected hallucinators and unselected non-hallucinators. The former group was stringently diagnosed and represented clear-cut cases of Mania, Schizophrenia, and Psychotic Depression. The second group of unselected patients was representative of the cases flowing through the neuropsychological laboratory at the Alberta Hospital Edmonton. This is a much more heterogeneous group and is therefore more representative of the kind of cases seen by hospital clinicians (Goldstein & Shelly, 1972).

While group differences were evident, it was not in terms of the symptom of interest, that is hallucinations, but rather differences were due to the effects of age, sex, and control versus patient group membership. The most probable reason for these findings is that at a cerebral level, hallucinations are too specific a symptom for this battery of neuropsychological tests to detect. Thus while the neuropsychological measures used in this investigation have considerable sensitivity to brain dysfunction, in terms of patient/control differences, they lack specificity within the impaired groups for the variable of interest (Fletcher, Fletcher, & Wagner, 1982).

Alternatively, findings can be explained within the concepts of multifinality and equifinality (Benjamin, 1982). Expressed psychologically, they suggest that multiple phenomenologies may result from a single etiology, and conversely, multiple etiologies may result in a single phenomenology. In the case of hallucinations, they can result from a wide variety of causes, ranging from sleep deprivation to alcohol toxicity. Conversely, hallucinations may be one symptom among many, as a result of brain injury. In order to determine a neuropsychological profile of hallucinations, it may be necessary to use subjects with a single etiology for their hallucinations. By using an etiologically heterogeneous sample of hallucinators neuropsychological correlates based on psychometric test data may have been washed out.

An attempt to enhance sensitivity to specific areas of dysfunction by means of rank ratio scores also proved disappointing. Ratios of tests thought to be differentially sensitive to right-left and anterior-posterior brain regions, were constructed. However no Scheffe contrasts by group reached significance at the p<.05 level.

As a limitation of the study it is noted that the presence of hallucinations was more clearly established in the selected group. For the unselected group, the presence of hallucinations was determined by reading the patient's case notes, or at case conference. Whereas for selected patients two psychiatrists had to agree on the presence of the symptom, hallucinations.

Within the selected group, the effects of diagnosis are confounded by combining the three diagnostic subgroups, Manics, Schizophrenics, and Psychotic Depressives and then separating them on the basis of presence or absence of hallucinations. Thus the overlapping of symptoms from the three groups may have acted to obscure the effects of hallucinations on the neuropsychological measures. In addition, the modality of hallucination may have had a confounding effect. All subjects in the investigation experienced auditory hallucinations, but many cases experienced hallucinations of other senses as well. All of the subjects in this study were hospitalized and the majority of unselected cases were medicated at the time of testing, whereas selected cases were largely unmedicated. The effects of psychotropic medications on neuropsychological variables are equivocal with evidence for both improved and deteriorated functioning present in the literature (Brumback & Staton, 1980; Donnelly, Nasrallah, & Wyatt, 1978; Fromm & Schopflocher, 1984; Heaton, & Crowley, 1981; Hymowitz & Spohn, 1980; Medalia, Gold, & Merriam, 1988).

Subjects were typically assessed early in their stay in hospital, since neuropsychological results are commonly used as part of the diagnostic process. However, neither severity of illness or chronicity was directly controlled for and patients may have had previous hospitalizations and thus been exposed to more medications as well as having had a longer history of illness and hence hallucinations.

6.3 Implications of Findings

Hallucinations have rarely been studied by means of a standardized neuropsychological test battery. Rather, different diagnostic groups incorporating hallucinations along with a myriad of other symptoms have been investigated (Flor-Henry, 1983a, 1983b, in press b; Flor-Henry, Fromm-Auch, & Schopflocher, 1983; Silverstein & Arzt, 1985; Silverstein, McDonald, & Meltzer, 1988; Silverstein & Zerwic, 1985). The purpose of this investigation was to evaluate the neuropsychological correlates of hallucinations as a symptom, independent of diagnosis.

This approach, using neuropsychological tests to assess "discrete" behavioral symptoms, rather than clusters of symptoms, that is diagnoses, has been strongly advocated by Persons(1986). She argues that assessment of symptoms avoids diagnostic misclassification due to symptom overlap, allows for the investigation of specific aspects of symptomatology, for example type of thought disorder, contributes to the development of empirically testable psychological theory, recognizes the continuous nature of symptomatic behavior from normal to pathological, and lastly, contributes to the refinement of nosological systems.

A similar approach has been advocated by Goldstein (1984, 1987), Miller (1985a), and Schear (1987). These authors stress the heterogeneity of neuropsychological results within traditional psychiatric groupings and emphasize the role of the task demand components that make up neurocognitive functions. For examples of this approach to the neuropsychological analysis of behavior see Johanson, Gustafson, and Risberg (1986) and Kiernan (1981). Classification schemes based on neuropsychological assessment of psychiatric patients, independent of diagnosis, have been developed by Townes et al. (1985) and Yozawitz (1986). They stress functional competency and relate their findings to treatment planning.

The essential point is that neuropsychological test patterns and traditional psychiatric diagnoses tend not to correlate very highly (Goldstein, 1987). Predictors, in this case neuropsychological tests, can do no better than the criteria, that is psychiatric diagnoses, that they purport to assess. This is not meant to imply that the process of diagnosis is a futile endeavor; in psychiatry it is unavoidable (Kendall, 1975). Rather it can be said that the behavioral domains sampled by neuropsychological tests and psychiatric diagnoses do not substantially overlap.

The current investigation failed to find a relationship between the target symptom, hallucinations, and neuropsychological test results for either heterogeneous psychiatric patients or strictly diagnosed cases. To the author's knowledge the only other study specifically investigating hallucinations by means of neuropsychological tests is that of McKay, Golden, and Scott (1981). These authors, using the Luria-Nebraska Neuropsychological Test Battery (Golden, Hammeke, & Purisch, 1980), found general cognitive impairment with a left frontal emphasis in the auditorily hallucinated subjects (n=10), no impairment in the visually hallucinated subjects (n=10), and an intermediate profile in the non-hallucinated group (n=20). The present study used a much larger sample, including a control group, a different test battery (expanded Halstead-Reitan battery), but did not match subjects for demographic, medication, or chronicity variables.

Not directly related to the purpose of the investigation, but relevant to the area of neuropsychological assessment of psychiatric patients are results of the supplementary correlational and factor analyses with this data set. Spearman Rho correlations are, generally, higher among the patients than the controls (.30 to .50 vs. .10 to .30 see Appendix I, Tables AI.1 and AI.2). Wiens and Matarazzo (1977) reported no significant correlations between WAIS Full Scale IQ and Halstead Reitan variables in a sample of 44 healthy, intelligent young males with a mean WAIS Full Scale IQ of 117.9. Inspection of Tables AI.1 and AI.2 shows that for patients the correlation between Verbal, Performance, and Full Scale IQ scores is much greater than for controls. This is not surprising in view of the wider range of IQ scores among the patient groups (see Table 5.1).

However, although intelligence is significantly related to neuropsychological test performance in this particular patient sample, less test variance is shared than is independent for the two sets of measures (cf. Moses, 1985 for similar results). This implies that the WAIS-R and neuropsychological variables are sensitive to different aspects of patient performance and are thus not redundant. These correlations and conclusions concerning the overlap of Wechsler and neuropsychological variables are consistent with published literature (Kupke & Lewis, 1985; Seidenberg, Glordani, Berent, & Boll, 1983; Warner, Ernst, Townes, Peel, & Preston, 1987).

In addition to Spearman Rho correlations, a canonical analyses was performed on the ranks. This showed that for controls 10% of WAIS-R variance was predictable from neuropsychological variables, while 4% of neuropsychological test variance was predictable from WAIS-R variables. For patients, 28% of the WAIS-R is predictable from neuropsychological variables, while 18% of neuropsychological test variance is predictable from the WAIS-R (see Table AI.3 and AI.4). A recent study by Zillmer, Fowler, Newman, and Archer (1988) with neuropsychiatric patients used regression analysis to predict 39% of the Halstead Reitan neuropsychological test battery variance from WAIS subtests. This is twenty-one percent higher than in this study, and may be due in part to differences in sample size, subject selection variables, and assessment procedures. Also the Sillmer, Fowler, Newman, and Archer (1988) data does not appear to have been ranked or treated nonparametrically, resulting in a possible spurious inflation of correlations and hence prediction. The authors note that their results are somewhat higher than those of previous studies and relate their findings to the fact that subjects with low cognitive abilities tend to do poorly on neuropsychological measures.

6.4 Directions for Further Research

Given the lack of substantive findings specific to the neuropsychological aspects of hallucinations in the present investigation, there are several areas into which future research could be profitably directed. These include the combination of neuropsychological with electrophysiological methods, such as computerized power spectral analysis and evoked potential assessment. Additionally, further investigation of the cognitive characteristics of hallucinators could prove useful. These potential directions for further research are discussed in the following sections.

One of the limitations of neuropsychological measures is their sensitivity to cortical over subcortical processes (Beaumont, 1983; Royce, Yeudall, & Bock, 1976). This is especially problematic in the context of West's (1962, 1968, 1975) arousal model of hallucinations, which stresses the role of the ascending reticular activating system (Barr & Kiernan, 1983). It is thus possible that hallucinatory activity so generated is "silent" in terms of neurocognitive sequelae, as assessed by neuropsychological tests. If hallucinations can result from disordered arousal mediated by brainstem circuitry the question arises as to why hallucinations are ordinarily modality specific, or at least limited to only one or two forms of sensory expression. Usually, hallucinations do not involve auditory, visual, tactile, olfactory and gustatory components simultaneously, but are typically limited to a single modality. It may be that cortically, mediated brain dysfunction has a crucial role in determining the way in which each individual experiences their own hallucinations. Therefore both cortical and subcortical brain systems may play a joint role in the genesis and expression of hallucinations.

If one accepts the idea that the contents of hallucinations are substantially derived from internal cerebral events, specifically traces of previously acquired memory, then this can be seen as providing a third level of brain system disturbance necessary for the hallucinatory experience. Firstly there is disturbed arousal mediated by the ascending reticular activating system, secondly dysfunction of higher level cortical brain systems, and thirdly disturbed hippocampal/limbic function, Both the amygdala and the hippocampus are likely to be involved in the process of hallucinating. The amygdala is thought to provide affective coloring to memories as well as play a role in cross-modal recall, while the hippocampus is directly implicated in recognition memory and learning of spatial relations (Mishkin & Appenzeller, 1987). The ascending reticular activating system projects to the thalamus and then to the cerebral cortex, where it has a substantial but nonspecific effect on consciousness (Barr & Kiernan, 1983). At a cortical level the temporal lobes are the most frequently disturbed region in organically based hallucinations. They are closely linked to the amygdala and hippocampus and function as processors of primarily auditory information (Lezak, 1983). The most common type of hallucinations are auditory. Thus hallucinations are conceptualized as cerebral events involving brain circuitry at multiple sites, including the brainstem, the amygdala, the hippocampus, the thalamus, and the cortex, particularly the temporal lobes.

In order to assess subcortical brain functions, electrophysiological techniques such as evoked potentials or computerized power spectral analysis of the EEG are necessary. Two forms of event related potentials could be of use in the subcortical assessment of hallucinations. These are brainstem auditory evoked potentials (Chiappa, 1985; Picton, Stapells, & Campbell, 1981; Shin, Tadashi, Soyer, Choi, Bonikowski, & Vitek, (1981); Starr, 1978) and the Galambos 40 Hz procedure which assesses midbrain/cortical connections (Galambos, Makeig, & Talmachoff, 1981; Makela, & Hari, 1987; Spydell, Pattee, & Goldie, 1985). Combined with neuropsychological assessment of the cortex, these techniques provide a comprehensive survey of brain function.

Relevant to this proposal is a recent study by Lindstrom, Klockhoff, Svedberg, and Bergstrom (1987). These authors recorded auditory brainstem responses (ABRs) in 20 schizophrenic inpatients assessed by the Research Diagnostic Criteria of Spitzer, Endicott, and Robins (1977). Abnormal responses were present in 10 out of the 20 cases. These did not correlate with degree of psychopathology, type of schizophrenia, medication history, age, sex, or degree of ventricular enlargement.

However, abnormal ABRs were present in 9 of the 11 schizophrenics admitting to auditory hallucinations, and in only 1 of the 9 patients who denied a history of hallucinations. The authors conclude that, "interference with the auditory pathways in the brain-stem may induce auditory hallucinations in schizophrenic patients" (Lindstrom, Klockhoff, Svedberg, & Bergstrom, 1987, p.9). Results of neuropsychological assessment would have been of great interest in these cases.

Another electrophysiological procedure, sensitive to cortical and subcortical events, that has been used to assess psychopathology (including hallucinations) is computerized power spectral analysis of the EEG. Following the studies of Galin and Ornstein (1972) who investigated lateralized cognitive activity in humans, varying forms of power spectral analysis have been used to investigate hallucinations in alcoholics (Spehr & Stemmler, 1985), drug induced hallucinations in normals (Koukkou & Lehmann, 1976), and hallucinations in schizophrenics (Heath, Walker, 1985; Whitton, Moldofsky, & Lue, 1978). No clear pattern of EEG sequelae of hallucinations has emerged, partly due to inconsistent methodologies (Serafetinides, Coger, & Martin, 1986). However, most studies agree in reporting abnormal power spectra during periods of overt psychotic behavior which typically include hallucinations (Stevens et al, 1979; Stevens & Livermore, 1982). The Heath and Walker (1985) study is of especial interest, because although it describes only two cases, the authors used depth as well as surface electrodes and reported subcortical abnormalities during hallucinations involving the hippocampus and geniculates. Evoked potential methods could provide independent confirmation of this kind of subcortical abnormality.

While the main thrust of this investigation has been neuropsychological, an important area for further research is that of the cognitive characteristics of hallucinators. Questionnaire assessment in this area has been conducted by several authors over the last few years (Bell, Billington, & Becker, 1985; Judkins & Slade, 1981; Junginger & Frame, 1985; Phillipson & Harris, 1985; Young, Bentall, Slade, & Dewey, 1986, 1987). Typically, the questionnaires are given to normal subjects, as well as to hallucinated and non-hallucinated psychiatric patients. Results of the studies are ambiguous with no clear cognitive pattern emerging, although the findings are in favor of conceptualizing hallucinations as being on a perceptual continuum that ranges from normal to abnormal sensory experience.

This is perhaps not surprising, since there is no reason to suppose that hallucinators, even within the "functional" psychoses, should be a homogeneous group. Consistent with this, Heilbrun and his colleagues (Heilbrun, 1980; Heilbrun & Blum, 1984; Heilbrun, Diller, Fleming & Slade, 1986) have studied DSM III diagnosed schizophrenics sorted according to the process/reactive continuum and suggested that each group has their own unique disattention strategy for coping with auditory hallucinations.

To summarize the preceding material, it has been suggested that electrophysiological methodologies such as evoked potentials and power spectral analysis be used in conjunction with neuropsychological tests to more fully assess all levels of the brain. Additionally, questionnaire assessment of the cognitive styles of different groups of hallucinators could provide psychological correlates of physiological measures. Each of these three modes of assessment have been used in isolation, but in order to more fully understand complex, multi-determined phenomena like hallucinations, it is necessary to integrate divergent forms of assessment on the same subjects. These views are consistent with the neuropsychological test design suggestions of Freides (1985), who argues for assessment based on an array of cognitive, perceptual, behavioral and psychophysiological measures. Ultimately, such an integrative approach should result in a better understanding of the etiology of hallucinations. This in turn should facilitate both the evaluation and implementation of more effective treatment strategies for hallucinations.

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Appendix I

Correlational Analyses

This appendix contains tables reporting Spearman Rho correlations for controls and patients between WAIS-R and neuropsychological variables (Tables AI.1, AI.2). Additionally, canonical correlations of WAIS-R and neuropsychological variables are reported for controls and patients (Tables AI.3, AI.4). The last two tables report on squared multiple correlations that predict WAIS-R subtest variance and neuropsychological variable variance for patients and controls (Tables AI.5, AI.6). See Table 4.1 for an explanation of the test abbreviations used in this appendix.

Table AI.1

Spearman Rho Correlations Between Neuropsychological and WAIS-R Variables for Controls (N=182)

Neuropsychological Variables	WAIS-R Variables													
	INFO	COMP	ARIT	SIMI	DSPA	VOCA	DSYM	PCOM	BDES	PARR	OASS	VIQ	PIQ	FSIQ
CPM	.13	.03	.19	.06	.09	.15	.06	.11	.13	.11	.1[illegible]	.17	.16	.17
OWF	.21	.31	.09	.17	.13	.28	.08	-.04	.12	-.08	.09	.19	.01	.15
PURDP	-.12	.05	-.07	-.01	.01	-.03	.20	-.05	.02	-.10	.06	-.10	.03	-.06
PURDNP	-.05	.09	-.03	-.08	-.03	-.01	.10	-.03	.00	-.04	-.06	-.08	-.01	-.06
PURDB	-.03	.08	-.03	.05	.01	-.01	.21	-.02	.15	-.10	.13	-.01	.09	.04
PURDA	-.18	.09	-.01	.04	.05	-.07	.27	-.02	.13	.02	.04	.00	.14	.07
LJPT	-.03	.07	.11	.07	.01	.01	-.07	.26	.16	.10	.19	.15	.33	.25
LJNPT	.05	.08	.12	.00	-.01	.00	.04	.20	.14	.13	.25	.11	.35	.24
LJBT	-.11	.05	.07	.02	.04	.00	.02	.27	.13	.19	.19	.10	.38	.26
WCST	.08	.21	.16	.21	.10	.20	.19	.10	.16	.15	.08	.23	.24	.29
WCSCAT	.00	.08	.18	.13	.16	.08	.08	.07	.19	.09	.12	.16	.22	.23
SPEECHS	.16	.18	.05	.03	.11	.21	.02	.23	.12	.00	.08	.13	.15	.15
TRAILA	-.02	-.03	.01	.05	.14	-.06	.22	.20	.14	.08	.08	.03	.27	.16
TRAILB	.05	.00	.17	.11	.19	.01	.25	.16	.21	.14	.03	.19	.29	.27
HALCAT	.28	.13	.28	.23	.23	.28	-.06	.22	.34	.13	.17	.39	.27	.40
FTAPP	.24	.22	.11	.19	.07	.26	.04	.40	.08	.09	.03	.19	.22	.22

Table AI.1 (continued)

Neuropsychological Variables	WAIS-R Variables													
	INFO	COMP	ARIT	SIMI	DSPA	VOCA	DSYM	PCOM	BDES	PARR	OASS	VIQ	PIQ	FSIQ
FTAPNP	.21	.18	.13	.17	.13	.24	.05	.33	-.01	.08	.03	.18	.16	.20
DYNP	.30	.10	.26	.03	.12	.20	-.17	.24	.10	.01	.00	.09	.02	.07
DYNNP	.24	.11	.27	.03	.10	.19	-.12	.18	.07	.01	.17	.10	.02	.07
SEAR	-.07	.02	.06	.09	.33	.13	.12	-.04	-.01	.07	.03	.16	.07	.13
FLOCPSS	-.01	.09	.01	-.07	.05	.03	-.04	.01	.07	.06	.12	.06	.04	.00
FLOCPDS	.04	-.08	.16	-.02	.14	.07	.08	.06	.10	.07	.04	.11	.10	.14
FLOCNPDS	-.07	-.01	.12	.01	.08	.01	.11	-.02	.04	.08	.09	.02	.08	.06
FLOCNPDS	.02	-.11	.10	.04	.14	.10	.24	.02	.09	.07	-.02	.07	.11	.11

Table AI.2

Spearman Rho Correlations Between Neuropsychological and WAIS-R Variables for Patients (N=161)

Neuropsychological Variables	WAIS-R Variables													
	INFO	COMP	ARIT	SIMI	DSPA	VOCA	DSYM	PCOM	BDES	PARR	OASS	VIQ	PIQ	FSIQ
CPM	.34	.37	.35	.42	.31	.44	.27	.36	.38	.32	.26	.48	.41	.48
OWF	.30	.47	.41	.36	.41	.53	.41	.29	.29	.12	.10	.53	.32	.50
PURDP	.20	.32	.32	.19	.23	.26	.35	.19	.25	.23	.13	.30	.27	.32
PURDNP	.18	.24	.28	.23	.22	.17	.37	.16	.25	.19	.11	.26	.27	.30
PURDB	.21	.25	.31	.19	.22	.25	.38	.12	.25	.15	.07	.29	.25	.32
PURDA	.11	.40	.37	.25	.26	.29	.58	.42	.38	.36	.18	.36	.46	.45
LJPT	.04	.04	.08	.03	.05	.04	.31	.18	.29	.14	.29	.04	.28	.16
LJNPT	-.04	.12	.05	.08	.02	.03	.30	.18	.32	.12	.36	.06	.30	.18
LJBT	.06	.13	.20	.16	.16	.08	.34	.28	.40	.18	.30	.17	.38	.29
WCST	.11	.23	.28	.19	.27	.27	.37	.40	.26	.30	.21	.28	.40	.38
WCSCAT	.16	.18	.24	.24	.20	.19	.30	.28	.23	.22	.25	.26	.30	.30
SPEECHS	.17	.36	.35	.39	.50	.37	.31	.38	.36	.29	.28	.45	.37	.47
TRAILA	.13	.32	.34	.23	.27	.24	.48	.46	.44	.43	.34	.36	.56	.49
TRAILB	.32	.43	.52	.39	.42	.39	.59	.49	.54	.39	.28	.52	.56	.60
HALCAT	.30	.30	.37	.33	.40	.35	.42	.47	.44	.43	.25	.43	.46	.49
FTAPP	.25	.28	.36	.24	.19	.31	.20	.33	.29	.32	.17	.37	.32	.39
FTAPNP	.19	.23	.30	.21	.17	.24	.16	.40	.36	.30	.24	.30	.36	.37

Table AI.2 (continued)

Neuropsychological Variables	WAIS-R Variables													
	INFO	COMP	ARIT	SIMI	DSPA	VOCA	DSYM	PCOM	BDES	PARR	OASS	VIQ	PIQ	FSIQ
DYNP	.14	.19	.24	.15	.04	.05	.10	.33	.36	.32	.30	.19	.35	.28
DYNNP	.09	.16	.23	.11	.01	.01	.08	.30	.31	.26	.23	.15	.28	.22
SEAR	.31	.46	.38	.38	.47	.49	.35	.43	.37	.21	.29	.49	.41	.53
FLOCPSS	.17	.23	.29	.32	.37	.29	.31	.24	.23	.15	.17	.35	.29	.35
FLOCPDS	.19	.25	.40	.25	.43	.27	.41	.37	.28	.16	.17	.37	.38	.43
FLONPSS	.19	.20	.34	.19	.42	.28	.34	.20	.17	.08	.08	.32	.23	.31
FLOCNPDS	.22	.25	.43	.24	.49	.31	.36	.32	.25	.15	.12	.38	.33	.41

Table AI.3

Canonical Correlations of WAIS-R with Neuropsychological Variables for Controls

Eigenvalue	Correlations	p Value	Redundancy Index WAIS-R	Redundancy Index Neuropsych.
.42	.65	.000	.10	.04
.39	.63	.059	.04	.03
.27	.52	.252	.03	.01
.23	.48	.550	.02	.01
.20	.45	.805	.02	.01
.18	.42	.950	.01	.01
.13	.36	.975	.01	.00
.09	.31	.974	.01	.01
.08	.28	.970	.01	.00
.06	.25	.932	.00	.00
.04	.21	NS	.00	.00

Note. Significance level (p value) is based on Bartlett's Test for remaining eigenvalues.

Table AI.4

Canonical Correlations of WAIS-R with
Neuropsychological Variables for Patients

Eigenvalue	Correlations	p Value	Redundancy Index WAIS-R	Redundancy Index Neuropsych.
.71	.84	.000	.28	.18
.46	.68	.000	.05	.03
.36	.60	.017	.02	.02
.29	.54	.122	.01	.02
.23	.48	.276	.02	.01
.21	.46	.507	.01	.01
.17	.41	.655	.01	.01
.14	.38	.757	.01	.01
.11	.33	.736	.01	.00
.10	.31	.727	.00	.00
.07	.27	NS	.00	.00

Note. Significance level (p value) is based on Bartlett's Test for remaining eigenvalues.

Table AI.5

Squared Multiple Correlations for Predicting WAIS-R From Remaining WAIS-R and Neuropsychological Variables for Patients and Controls

	Patients		Controls	
WAIS-R Variables	WAIS-R With WAIS-R R^2	WAIS-R with Neuropsych. R^2	WAIS-R with WAIS-R R^2	WAIS-R with Neuropsych. R^2
INFO	.57	.30	.47	.31
COMP	.59	.43	.29	.26
ARIT	.55	.40	.34	.23
SIMI	.51	.38	.21	.20
DSPA	.33	.47	.19	.22
VOCA	.66	.46	.51	.28
DSYM	.41	.54	.05	.26
PCOM	.47	.48	.15	.31
BDES	.53	.47	.22	.20
PARR	.42	.39	.14	.14
OASS	.39	.37	.21	.19

Table AI.6

Squared Multiple Correlations for Predicting Neuropsychological Variables from Remaining Neuropsychological and WAIS-R Variables for Patients and Controls

Neuropsych Variables	Patients		Controls	
	Neuropsych with Neuropsych.	Neuropsych. with WAIS-R	Neuropsych. with Neuropsych.	Neuropsych. with WAIS-R
	R^2	R^2	R^2	R^2
CPM	.42	.30	.11	.06
OWF	.46	.38	.11	.19
PURDP	.64	.13	.47	.09
PURDNP	.64	.18	.50	.05
PURDB	.73	.18	.47	.09
PURDA	.55	.42	.37	.17
LJPTA	.61	.21	.59	.12
LJNPT	.67	.24	.67	.11
LJB	.66	.25	.59	.17
WCST	.61	.24	.34	.15
WCSCAT	.58	.15	.26	.10
SPEECHS	.43	.36	.21	.11
TRAILA	.56	.37	.35	.13
TRAILB	.67	.47	.33	.15
HALCAT	.62	.42	.23	.22

Table AI.6 (Continued)

WAIS-R Variables	Patients		Controls	
	Neuropsych with Neuropsych.	Neuropsych. with WAIS-R	Neuropsych. with Neuropsych.	Neuropsych. with WAIS-R
	R^2	R^2	R^2	R^2
FTAPP	.65	.19	.74	.20
FTAPNP	.66	.21	.69	.16
DYNP	.91	.28	.88	.17
DYNNP	.91	.26	.86	.13
SEAR	.44	.37	.18	.16
FLOCPSS	.43	.20	.22	.05
FLOCPDS	.64	.31	.37	.05
FLOCNPSS	.48	.20	.21	.07
FLOCNPDS	.62	.34	.40	.10

APPENDIX II

COMPOSITE NEUROPSYCHOLOGICAL PROFILES

This appendix contains composite profiles for each of the five groups used in the investigation. These are, in order of presentation, unselected non-hallucinators, unselected hallucinators, selected non-hallucinators, selected hallucinators, and controls. Profiles are based on median rather than mean scores, although since they are norm referenced, values are treated as means. Subjects are coded as males because there were more males included in the study. These profiles are for illustrative purposes and provide the reader a visual display of what an average subject looks like in terms of neuropsychological variables.

At the level of clinical interpretation, group 1, the unselected non-hallucinators are characterized by mild levels of cerebral dysfunction. This involves the right hemisphere and implicates frontal cognitive and motor systems. Group 2, the unselected hallucinators, show mild to moderate levels of cerebral dysfunction. Frontal cognitive and motor systems are disturbed, as are the perceptual systems of the nondominant temporal lobe. Group 3, the selected non-hallucinators, shows moderate levels of brain dysfunction. This appears to be bifrontal in nature (right > left) and also involves the nondominant temporal lobe. Group 4, the unselected hallucinators, also shows moderate levels of cerebral dysfunction. This involves both hemispheres, primarily the temporal lobes, as well as the right frontal lobes (motor and cognitive). Group 5, the controls, have results that results are entirely within normal limits.

Clinically, there is an increase in the severity of dysfunction displayed in the profiles as one moves from group 1 through to group 4. Group 1 shows less dysfunction than group 2, group 2 less than group 3, and group 3 is similar to group 4. With increased disturbance comes greater temporal lobe disorganization, while parietally mediated sensory-perceptual systems are essentially unimpaired for all groups. Such pathology, as is being reflected by neuropsychological measures, is a function of anterior brain systems, that is, the frontal and temporal lobes. These configurally interpreted profiles are in essential agreement with the formal rank based analyses in terms of the increased severity of dysfunction dimension found across the four groups of patients.

COMPOSITE NEUROPSYCHOLOGICAL ABILITY PROFILE

UNSELECTED NON-HALLUCINATORS

```
                                        ADULT: BY SEX & AGE

                                        STANDARD DEVIATIONS :
                                        IMPAIRED     NORMAL  ABOVE NORMAL
VERBAL ABILITIES                       -3   -2   -1    0    1    2    3

                                        -------------------------------
ORAL WORD FLUENCY                 12.33 |    .    |X   .    |    .    |
SPEECH SOUNDS PERCEPTION           4.00 |    .    |    X    |    .    |
VERBAL IQ                         99.00 |    .    |    X    |    .    |
                                        -------------------------------
NONVERBAL ABILITIES

                                        -------------------------------
SEASHORE RHYTHM                    4.00 |    .   X|    .    |    .    |
COLOURED PROGRESSIVE MATRICES      2.00 |    .    |X   .    |    .    |
PERFORMANCE IQ                    96.00 |    .    |   X.    |    .    |
                                        -------------------------------
PERCEPTUAL-MOTOR ABILITIES I

                                        -------------------------------
TRAIL MAKING A                    25.40 |    .    |    X    |    .    |
TRAIL MAKING B                    59.00 |    .    | X  .    |    .    |
                                        -------------------------------
PERCEPTUAL-MOTOR ABILITIES II

                                        -------------------------------
PURDUE PEGBOARD: RIGHT            14.00 |    .   X|    .    |    .    |
PURDUE PEGBOARD: LEFT             13.00 |    .   X|    .    |    .    |
PURDUE PEGBOARD: BOTH             11.00 |    .   X|    .    |    .    |
PURDUE PEGBOARD: ASSEMBLIES       36.00 |    .    |X   .    |    .    |
                                        -------------------------------
ABSTRACT ABILITIES

                                        -------------------------------
CATEGORY TEST                     50.00 |    .  X |    .    |    .    |
WISCONSIN CARD SORT: ERRORS       19.00 |    .    |  X .    |    .    |
WISCONSIN CARD SORT: SUBTESTS      6.00 |    .    |   .X    |    .    |
                                        -------------------------------
LEARNING/MEMORY

                                        -------------------------------
                                        -------------------------------
ATTENTION/CONCENTRATION

                                        -------------------------------
SEASHORE RHYTHM                    4.00 |    .   X|    .    |    .    |
DIGIT SPAN                         9.00 |    .    |  X .    |    .    |
SPEECH SOUNDS PERCEPTION           4.00 |    .    |    X    |    .    |
                                        -------------------------------
ARITHMETIC ABILITY

                                        -------------------------------
ARITHMETIC                         9.00 |    .    |  X .    |    .    |
                                        -------------------------------
                                        STANDARD DEVIATIONS :
                                        IMPAIRED    NORMAL   ABOVE NORMAL
                                        -3  -2   -1    0    1    2    3
MOTOR FUNCTIONS

                                        -------------------------------
DYNAMOMETER: RIGHT                44.80 |    .    |X   .    |    .    |
FINGER TAPPING: RIGHT             48.40 |    .    |  X .    |    .    |
DYNAMOMETER: LEFT                 41.60 |    .    |X   .    |    .    |
FINGER TAPPING: LEFT              42.40 |    .    | X  .    |    .    |
                                        -------------------------------
SENSORY-PERCEPTUAL FUNCTIONS

                                        -------------------------------
FINGER LOCALIZATION: RIGHT         2.00 |    .    |    .X   |    .    |
L.J. TACTILE RECOG.: RIGHT        29.10 |    .    |    .X   |    .    |
FINGER LOCALIZATION: LEFT          3.00 |    .    |    X    |    .    |
L.J. TACTILE RECOG.: LEFT         27.90 |    .    |    X    |    .    |
L.J. TACTILE RECOG.: BOTH         21.80 |    .    |    .X   |    .    |
                                        -------------------------------
```

```
                                    STANDARD DEVIATIONS :
                                    IMPAIRED    NORMAL    ABOVE NORMAL
WAIS-R                             -3    -2    -1    0     1     2     3

INFORMATION                         -----------------------------------
DIGIT SPAN                   9.00  |     .     |   X .     |     .     |
VOCABULARY                   9.00  |     .     |   X .     |     .     |
ARITHMETIC                  10.00  |     .     |     X     |     .     |
COMPREHENSION                9.00  |     .     |   X .     |     .     |
SIMILARITIES                10.00  |     .     |     X     |     .     |
                            10.00  |     .     |     X     |     .     |
                                    -----------------------------------

                                    -----------------------------------
PICTURE COMPLETION          10.00  |     .     |     X     |     .     |
PICTURE ARRANGEMENT          9.00  |     .     |   X .     |     .     |
BLOCK DESIGN                10.00  |     .     |     X     |     .     |
OBJECT ASSEMBLY             10.00  |     .     |     X     |     .     |
DIGIT SYMBOL                 8.00  |     .     | X   .     |     .     |
                                    -----------------------------------

                                    -----------------------------------
VERBAL IQ                   99.00  |     .     |     X     |     .     |
PERFORMANCE IQ              96.00  |     .     |    X.     |     .     |
FULL SCALE IQ              100.00  |     .     |     X     |     .     |
                                    -----------------------------------
```

COMPOSITE NEUROPSYCHOLOGICAL ABILITY PROFILE

UNSELECTED HALLUCINATORS

```
                                         ADULT: BY SEX & AGE

                                         STANDARD DEVIATIONS :
                                         IMPAIRED     NORMAL  ABOVE NORMAL
VERBAL ABILITIES                        -3   -2   -1    0    1    2    3
                                         -------------------------------
ORAL WORD FLUENCY                  9.33  |    .X   |    .    |    .    |
SPEECH SOUNDS PERCEPTION           5.00  |    .    |  X .    |    .    |
VERBAL IQ                         91.00  |    .    | X  .    |    .    |
NONVERBAL ABILITIES                      -------------------------------

SEASHORE RHYTHM                          -------------------------------
COLOURED PROGRESSIVE MATRICES      5.00  |    .X   |    .    |    .    |
PERFORMANCE IQ                     3.00  |    . X  |    .    |    .    |
                                  86.00  |    .    X    .    |    .    |
PERCEPTUAL-MOTOR ABILITIES I             -------------------------------

TRAIL MAKING A                           -------------------------------
TRAIL MAKING B                    33.90  |    .   X|    .    |    .    |
                                  85.70  |   X.    |    .    |    .    |
PERCEPTUAL-MOTOR ABILITIES II            -------------------------------

PURDUE PEGBOARD: RIGHT                   -------------------------------
PURDUE PEGBOARD: LEFT             13.00  |    .X   |    .    |    .    |
PURDUE PEGBOARD: BOTH             12.00  |    .X   |    .    |    .    |
PURDUE PEGBOARD: ASSEMBLIES       10.00  |    X    |    .    |    .    |
                                  30.00  |X   .    |    .    |    .    |
ABSTRACT ABILITIES                       -------------------------------

CATEGORY TEST                            -------------------------------
WISCONSIN CARD SORT: ERRORS       76.00  X    .    |    .    |    .    |
WISCONSIN CARD SORT: SUBTESTS     37.00  |    .X   |    .    |    .    |
                                   5.00  |    .    |X   .    |    .    |
LEARNING/MEMORY                          -------------------------------

                                         -------------------------------
ATTENTION/CONCENTRATION                  -------------------------------

SEASHORE RHYTHM                          -------------------------------
DIGIT SPAN                         5.00  |    .X   |    .    |    .    |
SPEECH SOUNDS PERCEPTION           8.00  |    .    |X   .    |    .    |
                                   5.00  |    .    | X  .    |    .    |
ARITHMETIC ABILITY                       -------------------------------

ARITHMETIC                               -------------------------------
                                   7.00  |    .    X    .    |    .    |
                                         -------------------------------
                                         STANDARD DEVIATIONS :
                                         IMPAIRED    NORMAL   ABOVE NORMAL
MOTOR FUNCTIONS                        -3   -2   -1    0    1    2    3

                                        --------------------------------
DYNAMOMETER: RIGHT                34.00 |     X    |    .    |
FINGER TAPPING: RIGHT             47.00 |     .    | X  .   .|    .    |
DYNAMOMETER: LEFT                 33.30 |     . X  |    .    |    .    |
FINGER TAPPING: LEFT              41.40 |     .    |X   .    |    .    |
                                        --------------------------------
SENSORY-PERCEPTUAL FUNCTIONS

                                        --------------------------------
FINGER LOCALIZATION: RIGHT         4.00 |     .    | X  .    |    .    |
L.J. TACTILE RECOG.: RIGHT        36.10 |     .    |    X    |    .    |
FINGER LOCALIZATION: LEFT          6.00 |     .    X    .    |    .    |
L.J. TACTILE RECOG.: LEFT         39.20 |     .    | X  .    |    .    |
L.J. TACTILE RECOG.: BOTH         36.10 |     .    | X  .    |    .    |
                                        --------------------------------
```

```
                                         STANDARD DEVIATIONS :
                                         IMPAIRED    NORMAL   ABOVE NORMAL
WAIS-R                                  -3   -2   -1    0    1    2    3

                                         -------------------------------
INFORMATION                         7.00 |    .    X    .    |    .    |
DIGIT SPAN                          8.00 |    .    | X  .    |    .    |
VOCABULARY                          9.00 |    .    |  X .    |    .    |
ARITHMETIC                          7.00 |    .    X    .    |    .    |
COMPREHENSION                       7.00 |    .    X    .    |    .    |
SIMILARITIES                        8.00 |    .    | X  .    |    .    |
                                         -------------------------------

                                         -------------------------------
PICTURE COMPLETION                  8.00 |    .    | X  .    |    .    |
PICTURE ARRANGEMENT                 8.00 |    .    | X  .    |    .    |
BLOCK DESIGN                        8.00 |    .    | X  .    |    .    |
OBJECT ASSEMBLY                     8.00 |    .    | X  .    |    .    |
DIGIT SYMBOL                        6.00 |    . X  |    .    |    .    |
                                         -------------------------------

                                         -------------------------------
VERBAL IQ                          91.00 |    .    | X  .    |    .    |
PERFORMANCE IQ                     86.00 |    .    X    .    |    .    |
FULL SCALE IQ                      86.00 |    .    X    .    |    .    |
                                         -------------------------------
```

COMPOSITE NEUROPSYCHOLOGICAL ABILITY PROFILE

SELECTED NON-HALLUCINATORS

```
                                            ADULT: BY SEX & AGE

                                            STANDARD DEVIATIONS* :
                                            IMPAIRED     NORMAL  ABOVE NORMAL
                                           -3  -2  -1     0     1     2    3
VERBAL ABILITIES
                                          -------------------
ORAL WORD FLUENCY                   11.00 |  . X|  .  |  .  |
SPEECH SOUNDS PERCEPTION             7.00 |  .X |  .  |  .  |
VERBAL IQ                           93.00 |  .  | X.  |  .  |
                                          -------------------
NONVERBAL ABILITIES
                                          -------------------
SEASHORE RHYTHM                      6.00 | X.  |  .  |  .  |
COLOURED PROGRESSIVE MATRICES        3.00 |  .X |  .  |  .  |
PERFORMANCE IQ                      85.00 |  .  X  .  |  .  |
                                          -------------------
PERCEPTUAL-MOTOR ABILITIES I
                                          -------------------
TRAIL MAKING A                      33.90 |  . X|  .  |  .  |
TRAIL MAKING B                     102.40 X  .  |  .  |  .  |
                                          -------------------
PERCEPTUAL-MOTOR ABILITIES II
                                          -------------------
PURDUE PEGBOARD: RIGHT              14.00 |  . X|  .  |  .  |
PURDUE PEGBOARD: LEFT               13.00 |  . X|  .  |  .  |
PURDUE PEGBOARD: BOTH               11.00 |  . X|  .  |  .  |
PURDUE PEGBOARD: ASSEMBLIES         28.00 X  .  |  .  |  .  |
                                          -------------------
ABSTRACT ABILITIES
                                          -------------------
CATEGORY TEST                       83.00 X  .  |  .  |  .  |
WISCONSIN CARD SORT: ERRORS         41.00 |  X  |  .  |  .  |
WISCONSIN CARD SORT: SUBTESTS        6.00 |  .  |  .X |  .  |
                                          -------------------
LEARNING/MEMORY
                                          -------------------
                                          -------------------
ATTENTION/CONCENTRATION
                                          -------------------
SEASHORE RHYTHM                      6.00 | X.  |  .  |  .  |
DIGIT SPAN                           8.00 |  .  |X .  |  .  |
SPEECH SOUNDS PERCEPTION             7.00 |  . X|  .  |  .  |
                                          -------------------
ARITHMETIC ABILITY
                                          -------------------
ARITHMETIC                           9.00 |  .  |X .  |  .  |
                                          -------------------
```

```
                                        STANDARD DEVIATIONS :
                                        IMPAIRED    NORMAL   ABOVE NORMAL
MOTOR FUNCTIONS                        -3   -2   -1    0    1    2    3

                                        -------------------------------
DYNAMOMETER: RIGHT                34.10 |    X    |    .    |    .    |
FINGER TAPPING: RIGHT             44.80 |    .    |X   .    |    .    |
DYNAMOMETER: LEFT                 28.80 |   X.    |    .    |    .    |
FINGER TAPPING: LEFT              41.00 |    .    |X   .    |    .    |
                                        -------------------------------
SENSORY-PERCEPTUAL FUNCTIONS

                                        -------------------------------
FINGER LOCALIZATION: RIGHT         4.00 |    .    | X  .    |    .    |
L.J. TACTILE RECOG.: RIGHT        48.50 |    .    | X  .    |    .    |
FINGER LOCALIZATION: LEFT          8.00 |    .X   |    .    |    .    |
L.J. TACTILE RECOG.: LEFT         41.00 |    .    | X  .    |    .    |
L.J. TACTILE RECOG.: BOTH         43.40 |    .   X     .    |    .    |
                                        -------------------------------
```

```
                                         STANDARD DEVIATIONS :
                                         IMPAIRED    NORMAL   ABOVE NORMAL
WAIS-R                                 -3   -2   -1    0    1    2    3

                                         -------------------------------
INFORMATION                        10.00 |    .    |    X    |    .    |
DIGIT SPAN                          8.00 |    .    | X  .    |    .    |
VOCABULARY                          9.00 |    .    |  X .    |    .    |
ARITHMETIC                          9.00 |    .    |  X .    |    .    |
COMPREHENSION                       8.00 |    .    | X  .    |    .    |
SIMILARITIES                       10.00 |    .    |    X    |    .    |
                                         -------------------------------

                                         -------------------------------
PICTURE COMPLETION                  6.00 |    .  X |    .    |    .    |
PICTURE ARRANGEMENT                 7.00 |    .    X    .    |    .    |
BLOCK DESIGN                        9.00 |    .    |  X .    |    .    |
OBJECT ASSEMBLY                     9.00 |    .    |  X .    |    .    |
DIGIT SYMBOL                        6.00 |    .  X |    .    |    .    |
                                         -------------------------------

                                         -------------------------------
VERBAL IQ                          93.00 |    .    | X  .    |    .    |
PERFORMANCE IQ                     85.00 |    .    X    .    |    .    |
FULL SCALE IQ                      89.00 |    .    |X   .    |    .    |
                                         -------------------------------
```

COMPOSITE NEUROPSYCHOLOGICAL ABILITY PROFILE

SELECTED HALLUCINATORS

```
                                          ADULT: BY SEX & AGE

                                          STANDARD DEVIATIONS*:
                                          IMPAIRED     NORMAL  ABOVE NORMAL
VERBAL ABILITIES                         -3   -2   -1   0    1    2    3

                                          -------------------------------
ORAL WORD FLUENCY                   8.67  |   X.    |    .    |    .    |
SPEECH SOUNDS PERCEPTION            9.00  |  X .    |    .    |    .    |
VERBAL IQ                          88.00  |    .    |X   .    |    .    |
                                          -------------------------------
NONVERBAL ABILITIES
                                          -------------------------------
SEASHORE RHYTHM                     6.00  |    . X  |    .    |    .    |
COLOURED PROGRESSIVE MATRICES       4.00  |  X .    |    .    |    .    |
PERFORMANCE IQ                     82.00  |    .   X|    .    |    .    |
                                          -------------------------------
PERCEPTUAL-MOTOR ABILITIES I
                                          -------------------------------
TRAIL MAKING A                     41.10  |   X.    |    .    |    .    |
TRAIL MAKING B                     86.80  |    . X  |    .    |    .    |
                                          -------------------------------
PERCEPTUAL-MOTOR ABILITIES II
                                          -------------------------------
PURDUE PEGBOARD: RIGHT             15.00  |    .    |   X.    |    .    |
PURDUE PEGBOARD: LEFT              14.00  |    .    | X  .    |    .    |
PURDUE PEGBOARD: BOTH              12.00  |    .    | X  .    |    .    |
PURDUE PEGBOARD: ASSEMBLIES        27.00  |X   .    |    .    |    .    |
                                          -------------------------------
ABSTRACT ABILITIES
                                          -------------------------------
CATEGORY TEST                      85.00  X    .    |    .    |    .    |
WISCONSIN CARD SORT: ERRORS        33.00  |    .    X    .    |    .    |
WISCONSIN CARD SORT: SUBTESTS       6.00  |    .    |    .X   |    .    |
                                          -------------------------------
LEARNING/MEMORY
                                          -------------------------------
                                          -------------------------------
ATTENTION/CONCENTRATION
                                          -------------------------------
SEASHORE RHYTHM                     6.00  |    . X  |    .    |    .    |
DIGIT SPAN                          7.00  |    .    X    .    |    .    |
SPEECH SOUNDS PERCEPTION            9.00  |  X .    |    .    |    .    |
                                          -------------------------------
ARITHMETIC ABILITY
                                          -------------------------------
ARITHMETIC                          8.00  |    .    | X  .    |    .    |
                                          -------------------------------
```

```
                                             STANDARD DEVIATIONS :
                                             IMPAIRED    NORMAL    ABOVE NORMAL
                                            -3   -2   -1    0    1    2    3
MOTOR FUNCTIONS

                                             ------------------------------
DYNAMOMETER: RIGHT                    33.50 |    X    |    .    |    .    |
FINGER TAPPING: RIGHT                 38.00 |    .X   |    .    |    .    |
DYNAMOMETER: LEFT                     31.10 |   X.    |    .    |    .    |
FINGER TAPPING: LEFT                  36.20 |    . X  |    .    |    .    |
                                             ------------------------------
SENSORY-PERCEPTUAL FUNCTIONS

                                             ------------------------------
FINGER LOCALIZATION: RIGHT             5.00 |    .    X    .    |    .    |
L.J. TACTILE RECOG.: RIGHT            52.90 |    .    |X   .    |    .    |
FINGER LOCALIZATION: LEFT              5.00 |    .    | X  .    |    .    |
L.J. TACTILE RECOG.: LEFT             41.10 |    .    |   X.    |    .    |
L.J. TACTILE RECOG.: BOTH             31.80 |    .    | X  .    |    .    |
                                             ------------------------------
```

```
                                         STANDARD DEVIATIONS :
                                         IMPAIRED    NORMAL   ABOVE NORMAL
WAIS-R                                  -3   -2   -1    0    1    2    3
------
                                         -------------------------------
INFORMATION                         9.00 |    .    |  X .    |    .    |
DIGIT SPAN                          7.00 |    .    X    .   .|    .    |
VOCABULARY                          8.00 |    .    | X  .    |    .    |
ARITHMETIC                          8.00 |    .    | X  .    |    .    |
COMPREHENSION                       8.00 |    .    | X  .    |    .    |
SIMILARITIES                        8.00 |    .    | X  .    |    .    |
                                         -------------------------------

                                         -------------------------------
PICTURE COMPLETION                  6.00 |    .  X |    .    |    .    |
PICTURE ARRANGEMENT                 5.00 |    . X  |    .    |    .    |
BLOCK DESIGN                        8.00 |    .    | X  .    |    .    |
OBJECT ASSEMBLY                     5.00 |    . X  |    .    |    .    |
DIGIT SYMBOL                        8.00 |    .    | X  .    |    .    |
                                         -------------------------------

                                         -------------------------------
VERBAL IQ                          88.00 |    .    |X   .    |    .    |
PERFORMANCE IQ                     82.00 |    .   X|    .    |    .    |
FULL SCALE IQ                      84.00 |    .   X.    .    |    .    |
                                         -------------------------------
```

COMPOSITE NEUROPSYCHOLOGICAL ABILITY PROFILE

CONTROLS

```
                                        ADULT: BY SEX & AGE

                                        STANDARD DEVIATIONS* :
                                        IMPAIRED      NORMAL    ABOVE NORMAL
                                       -3    -2   -1     0     1     2     3
VERBAL ABILITIES
                                        -----------------------------------
ORAL WORD FLUENCY               14.00  |     .     |    X.     |     .     |
SPEECH SOUNDS PERCEPTION         4.00  |     .     |     X     |     .     |
VERBAL IQ                      113.00  |     .     |     .    X|     .     |
                                        -----------------------------------
NONVERBAL ABILITIES
                                        -----------------------------------
SEASHORE RHYTHM                  2.00  |     .     |     .X    |     .     |
COLOURED PROGRESSIVE MATRICES    1.00  |     .     |     X     |     .     |
PERFORMANCE IQ                 109.00  |     .     |     .   X |     .     |
                                        -----------------------------------
PERCEPTUAL-MOTOR ABILITIES I
                                        -----------------------------------
TRAIL MAKING A                  22.60  |     .     |     .X    |     .     |
TRAIL MAKING B                  48.10  |     .     |     .X    |     .     |
                                        -----------------------------------
PERCEPTUAL-MOTOR ABILITIES II
                                        -----------------------------------
PURDUE PEGBOARD: RIGHT          16.00  |     .     |     . X   |     .     |
PURDUE PEGBOARD: LEFT           16.00  |     .     |     . X   |     .     |
PURDUE PEGBOARD: BOTH           13.00  |     .     |     .X    |     .     |
PURDUE PEGBOARD: ASSEMBLIES     40.00  |     .     |     .X    |     .     |
                                        -----------------------------------
ABSTRACT ABILITIES
                                        -----------------------------------
CATEGORY TEST                   34.00  |     .     |     X     |     .     |
WISCONSIN CARD SORT: ERRORS      8.00  |     .     |     .X    |     .     |
WISCONSIN CARD SORT: SUBTESTS    6.00  |     .     |     .X    |     .     |
                                        -----------------------------------
LEARNING/MEMORY
                                        -----------------------------------
                                        -----------------------------------
ATTENTION/CONCENTRATION
                                        -----------------------------------
SEASHORE RHYTHM                  2.00  |     .     |     .X    |     .     |
DIGIT SPAN                      11.00  |     .     |     . X   |     .     |
SPEECH SOUNDS PERCEPTION         4.00  |     .     |     X     |     .     |
                                        -----------------------------------
ARITHMETIC ABILITY
                                        -----------------------------------
ARITHMETIC                      11.00  |     .     |     . X   |     .     |
                                        -----------------------------------
```

```
                                          STANDARD DEVIATIONS :
                                          IMPAIRED     NORMAL    ABOVE NORMAL
MOTOR FUNCTIONS                          -3    -2    -1    0    1    2    3

                                         ---------------------------------
DYNAMOMETER: RIGHT              39.00    |     .     X     .    |    .    |
FINGER TAPPING: RIGHT           49.00    |     .     |    X.    |    .    |
DYNAMOMETER: LEFT               35.90    |     .    X|     .    |    .    |
FINGER TAPPING: LEFT            45.20    |     .     |    X.    |    .    |
                                         ---------------------------------
SENSORY-PERCEPTUAL FUNCTIONS

                                         ---------------------------------
FINGER LOCALIZATION: RIGHT       2.00    |     .     |     .X   |    .    |
L.J. TACTILE RECOG.: RIGHT      29.00    |     .     |     . X  |    .    |
FINGER LOCALIZATION: LEFT        2.00    |     .     |     . X  |    .    |
L.J. TACTILE RECOG.: LEFT       24.00    |     .     |     .  X |    .    |
L.J. TACTILE RECOG.: BOTH       18.70    |     .     |     . X  |    .    |
                                         ---------------------------------
```

```
                                          STANDARD DEVIATIONS :
                                          IMPAIRED      NORMAL       ABOVE NORMAL
  WAIS-R                                 -3    -2    -1     0     1     2     3
                                          ------------------------------------
  INFORMATION                     12.00  |     .     |     .  X  |     .     |
  DIGIT SPAN                      11.00  |     .     |     . X   |     .     |
  VOCABULARY                      12.00  |     .     |     .  X  |     .     |
  ARITHMETIC                      11.00  |     .     |     . X   |     .     |
  COMPREHENSION                   11.00  |     .     |     . X   |     .     |
  SIMILARITIES                    11.00  |     .     |     . X   |     .     |
                                          ------------------------------------

                                          ------------------------------------
  PICTURE COMPLETION              11.00  |     .     |     . X   |     .     |
  PICTURE ARRANGEMENT             10.00  |     .     |     X     |     .     |
  BLOCK DESIGN                    12.00  |     .     |     .  X  |     .     |
  OBJECT ASSEMBLY                 11.00  |     .     |     . X   |     .     |
  DIGIT SYMBOL                    11.00  |     .     |     . X   |     .     |
                                          ------------------------------------

                                          ------------------------------------
  VERBAL IQ                      113.00  |     .     |     .    X|     .     |
  PERFORMANCE IQ                 109.00  |     .     |     .  X  |     .     |
  FULL SCALE IQ                  112.00  |     .     |     .    X|     .     |
                                          ------------------------------------
```